CANADIAN THESES ON MICROFICHE

I.S.B.N.

THESES CANADIENNES SUR MICROFICHE

National Library of Canada
Collections Development Branch

Canadian Theses on
Microfiche Service

Ottawa, Canada
K1A 0N4

Bibliothèque nationale du Canada
Direction du développement des collections

Service des thèses canadiennes
sur microfiche

## NOTICE

The quality of this microfiche is heavily dependent upon the quality of the original thesis submitted for microfilming. Every effort has been made to ensure the highest quality of reproduction possible.

If pages are missing, contact the university which granted the degree.

Some pages may have indistinct print especially if the original pages were typed with a poor typewriter ribbon or if the university sent us a poor photocopy.

Previously copyrighted materials (journal articles, published tests, etc.) are not filmed.

Reproduction in full or in part of this film is governed by the Canadian Copyright Act, R.S.C. 1970, c. C-30. Please read the authorization forms which accompany this thesis.

THIS DISSERTATION
HAS BEEN MICROFILMED
EXACTLY AS RECEIVED

## AVIS

La qualité de cette microfiche dépend grandement de la qualité de la thèse soumise au microfilmage. Nous avons tout fait pour assurer une qualité supérieure de reproduction.

S'il manque des pages, veuillez communiquer avec l'université qui a conféré le grade.

La qualité d'impression de certaines pages peut laisser à désirer, surtout si les pages originales ont été dactylographiées à l'aide d'un ruban usé ou si l'université nous a fait parvenir une photocopie de mauvaise qualité.

Les documents qui font déjà l'objet d'un droit d'auteur (articles de revue, examens publiés, etc.) ne sont pas microfilmés.

La reproduction, même partielle, de ce microfilm est soumise à la Loi canadienne sur le droit d'auteur, SRC 1970, c. C-30. Veuillez prendre connaissance des formules d'autorisation qui accompagnent cette thèse.

LA THÈSE A ÉTÉ
MICROFILMÉE TELLE QUE
NOUS L'AVONS REÇUE

NL-339 (r. 82/08)

Canada

0-315-05958-3

National Library of Canada | Bibliothèque nationale du Canada

Canadian Theses Division | Division des thèses canadiennes

Ottawa, Canada
K1A 0N4

53878

# PERMISSION TO MICROFILM — AUTORISATION DE MICROFILMER

• Please print or type — Écrire en lettres moulées ou dactylographier

Full Name of Author — Nom complet de l'auteur

Patrick Joseph CARNEY

Date of Birth — Date de naissance

March 17 1953

Country of Birth — Lieu de naissance

CANADA

Permanent Address — Résidence fixe

142 Galloway Drive
Sherwood Park, Alberta
T8A 2N2

Title of Thesis — Titre de la thèse

Psychophysiological Stress, Biofeedback, and Migraine Therapy

University — Université

U. of Alberta

Degree for which thesis was presented — Grade pour lequel cette thèse fut présentée

Ph.D.

Year this degree conferred — Année d'obtention de ce grade

1981

Name of Supervisor — Nom du directeur de thèse

Dr George Fitzsimmons

Permission is hereby granted to the NATIONAL LIBRARY OF CANADA to microfilm this thesis and to lend or sell copies of the film.

The author reserves other publication rights, and neither the thesis nor extensive extracts from it may be printed or otherwise reproduced without the author's written permission.

L'autorisation est, par la présente, accordée à la BIBLIOTHÈQUE NATIONALE DU CANADA de microfilmer cette thèse et de prêter ou de vendre des exemplaires du film.

L'auteur se réserve les autres droits de publication; ni la thèse ni de longs extraits de celle-ci ne doivent être imprimés ou autrement reproduits sans l'autorisation écrite de l'auteur.

Date

Sept, 2, 1981

Signature

Patrick Carney

NL-91 (4/77)

THE UNIVERSITY OF ALBERTA

# PSYCHOPHYSIOLOGICAL STRESS, BIOFEEDBACK, AND MIGRAINE THERAPY

by

© PATRICK CARNEY

A THESIS

SUBMITTED TO THE FACULTY OF GRADUATE STUDIES AND RESEARCH

IN PARTIAL FULFILMENT OF THE REQUIREMENTS FOR THE DEGREE

OF DOCTOR OF PHILOSOPHY

EDUCATIONAL PSYCHOLOGY

EDMONTON, ALBERTA

FALL, 1981

THE UNIVERSITY OF ALBERTA

**RELEASE FORM**

NAME OF AUTHOR PATRICK CARNEY

TITLE OF THESIS PSYCHOPHYSIOLOGICAL STRESS, BIOFEEDBACK, AND MIGRAINE THERAPY

DEGREE FOR WHICH THESIS WAS PRESENTED DOCTOR OF PHILOSOPHY

YEAR THIS DEGREE GRANTED FALL, 1981.

Permission is hereby granted to THE UNIVERSITY OF ALBERTA LIBRARY to reproduce single copies of this thesis and to lend or sell such copies for private, scholarly or scientific research purposes only.

The author reserves other publication rights, and neither the thesis nor extensive extracts from it may be printed or otherwise reproduced without the author's written permission.

(SIGNED) Patrick Carney

PERMANENT ADDRESS:

142 Galloway Dr.

Sherwood Park

T8A 2N2

DATED Sept. 3 1981

THE UNIVERSITY OF ALBERTA

FACULTY OF GRADUATE STUDIES AND RESEARCH

The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies and Research, for acceptance, a thesis entitled PSYCHOPHYSIOLOGICAL STRESS, BIOFEEDBACK, AND MIGRAINE THERAPY submitted by PATRICK CARNEY in partial fulfilment of the requirements for the degree of DOCTOR OF PHILOSOPHY.

Supervisor

External Examiner

Date. 20 July 1981

## Acknowledgements

The author would like to thank several people for their participation in this research:

Dr. George Fitzsimmons for stimulating my interest in the area of biofeedback and providing excellent supervision throughout my doctoral studies;

Dr. Bryan Hiebert for his time and interest in serving as External Examiner;

Dr. Bob Mulcahy for his advice and guidance in matters of psychophysiological measurement and statistics;

Dr. Robert Short for his comments and encouragement during my Master's and Doctoral Programs;

Dr. Norman Thomas for his enthusiastic input and interesting perspective;

The 60 volunteer subjects who put up with the many forms and other inconveniences in the course of their biofeedback training;

Finally, I would like to thank my wife Anne Louise and my daughter Erin whose support and understanding were the most important forms of feedback of all.

## Abstract

Muscle tension and vasomotor changes are implicated in the occurrence of migraine headaches. Both electromyographic (EMG) and digit temperature biofeedback training have been shown to reduce migraine pain. The following study was conducted to see whether EMG training and temperature training were differentially effective in reducing migraines among subjects who displayed differing patterns of psychophysiological reactivity to stress. Sixty migraine patients were divided into two groups on the basis of frontal EMG and digit temperature changes during a mental mathematics task. Those who demonstrated large EMG changes relative to temperature changes formed one group. Those who demonstrated large temperature changes relative to EMG changes formed the other group. Subjects from each group were randomly assigned to one of three treatment conditions: (a) EMG biofeedback, (b) digit temperature biofeedback, and (c) EMG and digit temperature biofeedback combined. In this manner the effectiveness of the three types of biofeedback training were compared and the efficacy of matching particular forms of biofeedback with differential patterns of psychophysiological reactivity was analysed.

A second analysis of the headache data was conducted in which subjects were reclassified as EMG or temperature responsive according to rates of physiological recovery to prestress levels rather than the amounts of physiological change. In this manner the efficacy of matching particular

forms of biofeedback with differential patterns of psychophysiological recovery was analysed.

All subjects were instructed to monitor headache intensity on an hourly basis for one month prior to treatment, during one month of treatment, and over a follow-up period of one month. Headaches were rated on a scale ranging from zero to five during each waking hour with zero indicating no headache and five indicating an intense, incapacitating headache. Treatment sessions were conducted for 20 minutes twice weekly with each subject over a four week period. In addition, at two months posttreatment a biofeedback booster session was conducted in an effort to make results more durable. All subjects were instructed to practice the relaxation skills learned in the laboratory twice daily for 15 minutes in their home.

Each subject's headache data were averaged to obtain a mean headache rating per day for each period including baseline, treatment, and follow-up. In the first analysis where subjects were grouped according to psychophysiological reactivity a significant reduction in migraines ($F(1, 42) = 13.29$; $p<.001$) was obtained, but clinical improvement was not differentiated among the two groups by three treatments combinations. A significant interaction among groups and treatments ($F(2, 49) = 3.92$; $p<.05$) did result when subjects were grouped according to psychophysiological recovery from stress.

Reduction in migraine ratings from baseline to follow-up ranged from 33% to 42% among the three treatment groups. A criterion of clinical improvement of at least 50% reduction in mean headache activity indicated that 21 out of 55 subjects benefited to this degree. Hours of daily headache activity rated as "very severe" (assigned a weight of four or five on the headache scale) were also considered and it was found that 32 out of 55 subjects benefited by the criterion of a 50% reduction.

## Table of Contents


| Chapter | | Page |
|---|---|---|
| I. | INTRODUCTION | 1 |
| | A. Overview | 2 |
| | Purpose | 4 |
| II. | REVIEW OF THE LITERATURE | 6 |
| | A. Therapeutic Intent | 6 |
| | Placebo Effects | 8 |
| | Nonspecific Treatment Factors In Biofeedback Therapy | 12 |
| | The Conditioned Response Model of Nonspecific Treatment Effects | 16 |
| | B. Stress and the Migraine | 17 |
| | C. Temperature Training Biofeedback | 22 |
| | Anecdotal Case Reports | 25 |
| | Systematic Case Studies | 26 |
| | Controlled Group Outcome Studies | 27 |
| | D. Biofeedback, Relaxation and Response Stereotypy | 31 |
| | E. Summary | 42 |
| | F. Research Questions | 43 |
| III. | METHOD | 45 |
| | A. Subjects | 45 |
| | B. Research Design | 49 |
| | Part 1 | 49 |
| | Part 2 | 50 |
| | C. Apparatus and Facilities | 51 |

D. Stress Profile Procedures .....53

Differential Reactivity Calculations .....53

E. Treatment Procedure .....55

Group 1: EMG Biofeedback Training .....55

Group 2: Digit Temperature Biofeedback Training .....56

Group 3: EMG and Digit Temperature Biofeedback Combined .....57

Follow-Up .....57

F. Dependent Measures .....57

G. Ethical Considerations .....58

IV. RESULTS .....59

A. Treatment Effects and Psychophysiological Reactivity .....59

Stress Profile Data .....59

Summary .....61

Daily Headache Record .....62

Summary .....64

B. Treatment Effects and Psychophysiological Recovery .....65

Stress Profile Reclassification .....65

Summary .....68

Daily Headache Record .....68

Summary .....72

C. Biofeedback Training Data .....73

Reactivity Groupings .....73

Recovery Groupings .....76

V. Discussion .....103

Major Findings .....103

The Research Questions .....106
Treatment Mechanisms .....111
REFERENCE NOTES .....114
REFERENCES .....115
APPENDIX A .....122
APPENDIX B .....124
APPENDIX C .....125
APPENDIX D .....127
APPENDIX E .....131
APPENDIX F .....134
APPENDIX G .....136

## List of Tables


| Table | | Page |
|---|---|---|
| 1 | Characteristics of Study Population | 47 |
| 2 | Mean Headache Levels Across Reactivity Groups, Treatments and Periods | 63 |
| 3 | Mean Headache Levels Across Responsivity Groups, Treatments and Periods | 70 |

## List of Figures


| Figure | | Page |
|---|---|---|
| 1 | Reactivity T Scores | 79 |
| 2 | Mean EMG Levels of the Two Stress Reactivity Groups During Stress Profile Phases | 81 |
| 3 | Mean Temperature Levels of the Two Stress Reactivity Groups During Stress Profile Phases | 83 |
| 4 | Diagram for Experimental Design | 85 |
| 5 | Mean Headache Intensity Levels for the Three Treatments, Two Groups, and Three Periods | 87 |
| 6 | Mean EMG Levels for the Two Reclassified Stress Responsivity Groups During Stress Profile Phases | 89 |
| 7 | Mean Temperature Levels for the Two Reclassified Stress Responsivity Groups During Stress Profile Phases | 91 |
| 8 | Mean Headache Intensity Levels for the Three Treatments, Two Reclassified Groups, and Three Periods | 93 |
| 9 | Mean Headache Reduction Scores from Pretreatment to Posttreatment for the Three Treatments and Two Reclassification Groups | 95 |
| 10 | Mean EMG Levels for the Three Treatment Groups During Stress Profile and Treatment Sessions | 97 |
| 11 | Mean Temperature Levels for the Three Treatment Groups During Stress Profile and Treatment Sessions | 99 |
| 12 | Mean EMG Levels for the Three Treatments and the Two Responsivity Groups During Stress Profile and Treatment Sessions | 101 |
| 13 | Mean Temperature Levels for the Three Treatments and the Two Responsivity Groups During Stress Profile and Treatment Sessions | 103 |

# I. INTRODUCTION

*"A wise man ought to realize that health is his most valuable possession and learn how to treat his own illnesses by his own judgement."* *Hippocrates*

Migraine headache is a common disorder which man has tolerated for centuries in the absence of any highly effective medicinal treatment (Schiller, 1975). Within the past decade there has been a vigorous interest in the treatment of migraines via biofeedback learning. The catalyst for this interest appears to have been the observation made by Sargent, Green, and Walters (1972) where in the course of training one female subject with electroencephalograph (EEG), electromyograph (EMG), and hand temperature biofeedback, the subject reported a spontaneous recovery from a migraine headache.

Numerous published reports suggest that various combinations of EMG muscle relaxation, autogenic training, Jacobsonian relaxation, and hand-warming biofeedback are effective in the reduction of migraine episodes. To date the critical components of the various treatment packages have not been identified. Furthermore, there has been no reported analysis of the efficacy of matching particular forms of biofeedback with the particular patterns of psycho-physiological reactivity that different individuals display. Different psychophysiological reactions to stress among

migrainous individuals may be an important source of variability with respect to the success or failure of any one form of biofeedback learning in alleviating the headaches.

A. Overview

Each of the three treatment procedures used in the present study involved biofeedback training in order to teach the subjects how to recognize physiological cues of stress and relaxation, and how to relax more effectively than they were already able to do. "Biofeedback refers to any technique which uses instrumentation to give a person immediate and continuing signals on change in a bodily function that he is not usually conscious of (Sullivan, 1975, p. 38)." Through regular practice of the relaxation skills being taught, it was hoped that subjects would be able to reduce the frequency of physiological stress and thereby reduce the frequency of migraine headaches which are believed to be stress induced (Henryk-Gutt & Rees, 1972; Kundrow, 1978).

This study incorporated a three (treatments) by two (groups) repeated measures across treatment phases format. On the basis of physiological reactivity to the stress of performing a mental arithmetic task, 60 migraine sufferers were divided into one of two groups: (a) *EMG Reactive*- relatively high EMG reactivity with relatively low temperature reactivity, and (b) *Temperature Reactive*-

relatively high temperature reactivity (hands cool) with relatively low EMG reactivity. Subjects from each group were then randomly assigned among three treatment groups: (a) EMG biofeedback, (b) hand temperature biofeedback, and (c) EMG and hand temperature biofeedback combined. In this manner the effectiveness of the three types of biofeedback training was compared and the efficacy of matching particular forms of biofeedback with particular forms of psychophysiological reactivity was analyzed.

A second analysis of the headache data was also conducted in which subjects were reclassified as EMG or temperature responsive according to rates of recovery from stress rather than the amounts of physiological change from prestress levels. In this manner the efficacy of matching particular forms of biofeedback with differential patterns of psychophysiological recovery was analysed.

All subjects were instructed to monitor headache intensity and duration daily for one month prior to treatment, during one month of treatment, and over a follow-up period of one year. Treatment sessions were conducted twice weekly with each subject over a four week period. In addition a two-month biofeedback follow-up session was conducted to assess the durability of physiological control. This follow-up was also used as a booster session in which subjects were given an opportunity to practice what they had learned during treatment. Hand temperature and EMG levels were monitored in all subjects

throughout treatment.

**Purpose**

The major hypothesis being tested in this study was that migraine patients who demonstrate differential psychophysiological stress reactivity in terms of EMG and hand temperature, would benefit differentially from EMG and temperature biofeedback treatments. More specifically it was hypothesized that subjects who demonstrate relatively more muscle tension than hand cooling to stress would achieve more headache reduction through EMG biofeedback, which monitors this sensitive system, than through temperature biofeedback. Conversely it was hypothesized that subjects who demonstrate relatively more hand cooling than muscle tensing when stressed, would achieve more headache reduction through hand temperature biofeedback.

Migraine headache is generally considered to be a cerebral-vascular phenomenon (Dalessio, 1972). However, the implicated vasomotor changes that occur with stress are part of a more general physiological stress response that includes additional components of the autonomic nervous system as well as the skeletal muscular system. Research evidence indicates that various forms of relaxation training will reduce migraines (Blanchard & Epstein, 1976; Lake, Rainey & Papsdorf, 1979). Given the fact that idiosyncratic profiles of psychophysiological stress are found among different individuals the author was led to hypothesize that each form of biofeedback-assisted relaxation, EMG and

temperature, would prove more beneficial to some persons than to others.

The three (treatments) by two (groups) repeated measures design used in the present study addressed the question of whether or not subjects learned to reduce migraines more effectively, when their treatment format matched their dominant form of physiological reactivity, without the need of a specific control group. Although all subjects received treatment for migraines it was hypothesized that some subjects would obtain more effective results than others. The combined EMG and hand-warming biofeedback group served as a double check on the efficacy of matching treatments to patterns of responsivity. In the combined group both EMG reactive subjects and temperature reactive subjects received training relevant to their more stress sensitive physiological system. Hence the design also tested the efficacy of using a combined EMG and temperature biofeedback approach versus using EMG or temperature training alone.

## II. REVIEW OF THE LITERATURE

### A. Therapeutic Intent

In the general fields of medical and psychological health, researchers are frequently called upon to evaluate the effectiveness of particular treatment regimes used for particular health disorders. Clinical treatment investigations in medicine and psychology must always deal with the fact that some subset of patients will likely demonstrate a significant therapeutic response in any clinical study having therapeutic intent, regardless of the treatment which was employed. Hence the researcher must demonstrate that an observed clinical improvement "goes beyond" that which is produced by therapeutic intent alone. If this is accomplished then it further behooves the researcher to demonstrate "how" the treatment works to produce the observed effects.

In recent years a new approach to health has gained interest in North America. The approach is referred to as behavioural medicine and it amounts to an increased awareness of important psychological variables in the treatment of disorders traditionally recognized as the domain of medicine. According to Schwartz and Weiss (1978) "behavioural medicine is the interdisciplinary field concerned with the development and integration of behavioural and biomedical science knowledge and techniques relevant to health and illness and the application of this

knowledge and these techniques in prevention, diagnosis, treatment and rehabilitation." It has come to be realized that the patient's beliefs, attitudes, behaviours and general life style play a role in any recovery-from-illness process. Medicinal treatment effects which medicine has been unable to explain in terms of biochemical action have always been labelled and shelved as "placebo" effects. Now, however, psychologists and physicians are demonstrating a desire to harness the placebo and to discover its mechanics so that its benefits may be optimized.

As yet the mechanism for the placebo is far from being understood. A concensus does exist that the placebo represents the brain's capacity to foster beneficial physiological changes in the body to promote healing. It is not surprising then that many psychologists and physicians within the behavioural medicine area have begun to use biofeedback approaches to treating illness. The use of biofeedback involves teaching a patient to make physiological changes in his body in order to promote healing. Biofeedback applications for a number of medical disorders have met with considerable success. However, a new dilemma has been created. Biofeedback treatments, like drug treatments may be subject to positive outcome effects which are not specifically related to the physiological changes aimed at by the biofeedback treatment.

The likelihood of nonspecific treatment effects occurring in biofeedback research makes it necessary for the

researcher to be wary of the possibility of falsely attributing positive outcomes to therapeutic procedures per se. This section on Therapeutic Intent is included at the outset of the literature review in order to put biofeedback treatment effects into perspective with respect to nonspecific treatment effects which have been found in biofeedback research and elsewhere. In the next few pages a brief overview of the literature on placebo effects found in drug therapy will be presented. Next the literature on nonspecific treatment factors in the area of clinical biofeedback will be discussed. Lastly, a conditioned response model of nonspecific treatment effects will be considered.

**Placebo Effects**

In medicine nonspecific treatment factors are referred to as the "placebo." A psychiatric dictionary (Hirsie & Campbell, 1960) defines placebo as "any medication (treatment) used to alleviate symptoms, not by reasons of specific pharmacologic action, but solely by reinforcing the patient's favorable expectancies from treatment." Patent medicine in North America was founded on the placebo effect when in 1796 Samuel Lee Jr. of Windham, Connecticut patented the first American medicine called Bilious Pills (Koenig, 1974). Lee's Bilious Pills were claimed to be a remedy for yellow fever, jaundice, dysentery, dropsy, worms, female complaints and biliousness. Despite the fact that these pills were pharmacologically ineffective except as a

purgative agent, millions were sold. In the late 19th century druggists in Germany began to separate chemically active medicinal agents from chemically inert ones. Since then drug companies have sustained a multi-million dollar industry convincing people to take medicines that contain such chemically active ingredients in order to relieve every type of pain or discomfort imaginable. Whether people generally get relief through the pharmacological action of their medicine is a moot point; that they believe they will get some relief is unquestionable.

Various explanations exist for how the placebo effect works. Perhaps the most common rationale is that a placebo pill changes a person's reaction to a pain sensation without actually altering the sensation. In other words the biochemical or physiological bases to the pain remain unchanged while the person becomes cognitively distracted or somehow unattentive to the pain sensation. Such diminished attention to the pain may lead the person to conclude that the pain has reduced. Another explanation for the placebo effect is that the suggestion that relief is imminent subsequent to taking medication results in the person becoming less physiologically aroused due to the cognitive anticipation of relief. Physiological arousal as a manifestation of anxiety is implicated in the experience of pain (Melzack, 1973). Attention, arousal and suggestion are considered to be important factors in the manifestation of pain. Each of these elements are highly influenced by the

cognitive domain.

The extent to which nonspecific or "placebo" factors can operate in treatment might be described as awesome. The proportion of patients in various studies who obtain drug-placebo relief to about one half of the original intensity of severe clinical pain is remarkably constant - about one-third (Fields, 1978; Wickramasekera, 1980). Shapiro's (1964) review of the placebo literature documents the potential of such effects in virtually every therapeutic area including "organic illnesses and incurable malignancies." Franks (1961) examined the area of psychotherapy with respect to suggestion and remarked that such effects were not even limited to bringing about death as evidenced by the practice of voodoo.

The review of the placebo literature by Wickramesekera (1980) led him to formulate the following conclusions:

(1) A subset of patients show a significant therapeutic response to "inert" or "placebo" substances, procedures, and objects in any clinical study.

(2) No reliable procedure exists to date to identify in advance the above subset of patients.

(3) The same subset may not reliably respond to placebo.

(4) Any object or procedure offered with therapeutic intent can under the "right" conditions generate placebo effects.

(5) The mechanism of the effect is unknown and all the "right" conditions are unclear (Wickramesekera, 1980; pg. 5).

Wickramasekera (1980) surmised that the placebo effect is a real effect that clinicians have regarded as a nuisance because:

(1) Its action is not logically related to the known etiology of the disease or condition.

(2) The mechanism of its action is unknown.

(3) The effect is unreliable.

(4) The effect is not durable.

(5) It is an effect that can occur in any therapeutic situation (Wickramasekera, 1980; pg. 5).

An experiment by Sternback (1964) on the physiological specificity of the placebo phenomenon is particularly relevant to nonspecific effects in medical treatments. He gave identical pills to three sets of subjects but varied the instructions about the effects that the pills would produce. Unknown to the subjects each pill consisted of a plastic-coated magnet which could be used to monitor gastrointestinal activity. The instructions for one group was that the pill would relax the stomach; for another group he stated that the pill was a stimulant to the stomach; for the third group he stated that the pill was a placebo and would have no effect. The results of the experiment showed that stomach motility varied in relation to the instructions given for most of the subjects in the study.

## Nonspecific Treatment Factors In Biofeedback Therapy

In psychology, as in medicine, it is widely appreciated that any successful therapeutic outcome obtained with a patient will to some extent be due to variables which are additional to those actually specified in the rationale for the treatment being used. In clinical research, control groups are frequently employed in order to ascertain the extent to which therapeutic outcome from a particular treatment variable goes beyond results obtained without this component variable being included in the treatment. An obvious shortcoming of outcome analysis using control groups is that while a particular treatment component may be shown to be important, one can only hypothesize or speculate about how this component operates to be effective. The operation of the processes responsible for change require a separate analysis. It must be shown that specific therapeutic components actively operate and contribute to final behaviour changes (outcomes) which are obtained. Without such an analysis it is always possible that a particular treatment variable is effective for reasons completely unrelated to the researcher's logic for including the variable in the treatment.

Kazdin (1979) has defined nonspecific treatment factors as unspecified variables included in treatment that are not unique to the technique under investigation. Such factors contribute to change in an uncontrolled manner and they may extend themselves across various treatment techniques.

Examples of nonspecific factors may be treatment credibility and expectancy for improvement which various treatment procedures might enhance (Kazdin, 1979). It behooves the researcher to try to control for such factors as expectancy and credibility. However, more research is needed in order to identify additional factors which may influence outcome. Wickramasekera (1980) points out that the effects are termed "nonspecific" because we do not know about their parameters and thus we are unable to manipulate such effects systematically.

Stroebel (Stroebel & Glueck, 1973) has coined the term "ultimate placebo" to describe biofeedback procedures as a means by which the patient himself and the placebo effect are put in a position of importance in suppressing illness. Stroebel is himself a physician, and he uses the term placebo here in a more general sense than the dictionary definition of placebo which was presented earlier. What he means is that in biofeedback the patients are not given a cure, rather, they are given a technique. They are responsible to practice this technique. Biofeedback may get its potency for changes in health by helping the patient experiment with an improved pace of living, patterns of thinking, body processes, behavioural habits and perceptual style. In this sense biofeedback indirectly enables the patients to decrease their susceptibility to illness by altering these dimensions of mind/body stress. Active effects of biofeedback control may be proportionately less

important to improved health than individualized lifestyle changes fostered through the biofeedback self-responsibility treatment philosophy (Stroebel & Glueck, 1973).

Andrasik and Holroyd (1980) conducted a controlled study to test specific and nonspecific effects in the treatment of tension headaches via biofeedback. Subjects were assigned to one of three treatments designed to produce either decreased, stable, or increased frontal EMG levels. However, all treatment subjects were instructed that they were learning to reduce frontal muscle tension. A fourth group was also included who recorded headaches but did not receive any form of treatment. Results of this study indicated that the three biofeedback procedures produced similar decreases in headache at both posttreatment and six week follow-up assessments. EMG data indicated that frontal muscle tension levels varied as intended in the design. The authors interpreted their results to suggest that actual learned reductions of frontal EMG activity may have played only a minor role in the biofeedback treatment of tension headaches, while nonspecific factors associated with biofeedback may have accounted for a significant proportion of the obtained outcomes.

In the Andrasik and Holroyd (1980) study a number of basic treatment elements were standardized among treatments which could conceivably contribute to treatment effects in many biofeedback treatment studies of headache. The variables included (a) headache monitoring, (b) a muscle

contraction explanation of headache, (c) a treatment rationale stressing the application of biofeedback, (d) specific suggestions and demands for improvement, (e) verbal reinforcement for improvement in biofeedback control and headache intensity and, (f) progressively shaped feedback to ensure success at learning the biofeedback task. These elements taken together may have enhanced the treatment variables of credibility and expectancy for improvement as discussed by Kazdin (1979). However, Andrasik and Holroyd (1980), Holroyd (1979), and Holroyd and Andrasik (1978) have argued that positive biofeedback effects may result largely through changes in person-environment interactions which are fostered by biofeedback treatment procedures. Biofeedback treatment procedures may be effective by teaching subjects to monitor the insidious onset of headache symptoms and influencing them to engage in cognitive and behavioural coping strategies that help to suppress headache occurrences. Perhaps treatment credibility and expectancy for improvement play a role in motivating subjects to engage in such coping responses. Furthermore, it seems likely that biofeedback training might help subjects recognize stress in their body whether or not they are able to achieve direct physiological control of the system being monitored. Desired physiological changes subsequent to becoming aware of somatic stress can be obtained through coping behaviours such as leaving a stressful situation or laying down for a rest.

## The Conditioned Response Model of Nonspecific Treatment Effects

A novel approach to understanding the placebo effect has recently been proposed by Wickramasekera (1980). This author has postulated a conditioned response model in which classical and instrumental learning paradigms are used to explain how neutral stimuli can acquire the ability to produce placebo effects through association with active ingredients. According to the model the neutral stimuli may be places, objects, procedures or persons; active ingredients may include powerful drug effects or specific treatment effects in psychotherapy. Wickramasekera predicts from his model that therapists who routinely employ active ingredients will get stronger placebo effects than therapists who routinely use "inert" ingredients. Hence, placebo effects which are conditioned responses will consist of anticipatory fractional components of the unconditioned response that the active ingredients produce. Furthermore, the continued existence of placebo effects depends on the continued existence and pairing of the active ingredients with the conditioned stimuli.

In Wickramasekera's model credibility as an influential force on outcome is explained in terms of attention and arousal. Socially learned cues for credibility serve as discriminative stimuli to optimize attention and arousal conditions for learning. According to Wickramasekera (1980) the dimensions of credibility stimuli might include such

factors as:

a) Professional title of the therapist (e.g. psychologist, doctor);

b) the impressiveness of the therapeutic setting (e.g., office, laboratory);

c)the impressiveness of the placebo per se (e.g., size of pill, dials on instrumentation);

d) the credibility of the procedural ritual (e.g., injection, repeating mantra, watching biofeedback signal) and;

e) positive aspects of the therapeutic relationship (e.g., unconditional positive regard, sincerity).

The discussion of placebo and nonspecific treatment effects which has been presented makes it clear that any research involving therapeutic intent must control for the possibility that positive outcomes may occur which are unrelated to the treatment procedures per se.

## B. Stress and the Migraine

Migraine headache is commonly described as recurrent episodes of throbbing head pain, usually unilateral in onset. In the migraine headache syndrome the head pain is frequently associated with irritability and nausea, as well as less common symptoms which include photophobia, vomiting, constipation, and diarrhea (Dalessio, 1972).

Three epidemological surveys that have been conducted by Waters and O'Connor (1975) found that migraine occurred

in 15 to 20 percent of the men sampled and in 23 to 29 percent of the women. According to Dalessio (1972), variability in periodicity is a characteristic feature of the syndrome. Apparently attacks may range from a few times per week to three or four migraines in a lifetime. Erratic patterns in some patients consist of frequent attacks for about a week every three or four months during twenty years or more. Still other patients have migraines once or twice a week for a period of less than a year and then they are free of attacks for several years in a row before they return.

The sites of the migraine headache are variable both within and among patients. Patients have indicated pain in the temporal, supra-orbital, frontal, retrobulbar, parietal, postauricular, and occipital regions. It is common for unilateral pain to vary from side to side in successive attacks. Furthermore, it is often the case that unilateral headaches become generalized during an attack (Dalessio, 1972).

Migraine headaches have been classified into five major categories: (a) classic, (b) common, (c) cluster, (d) hemiplegic and ophthalmoplegic, and (e) lower half (Ad Hoc Committee on the Classification of Headache, 1962). Two of these categories - classic and common, are relevant to the present study. In classic migraine the headache is preceded by prodromal symptoms consisting of well defined visual disturbances and sometimes other sensory or motor disturbances. Common migraine differs from the classic

migraine primarily because of the absence of prodromal symptoms and the fact that the pain is less often unilateral. Variables such as menstruation, occupation, and environment often appear related to the onset of this form of migraine (Dalessio, 1972). Classic and common migraines are often accompanied by a muscle contraction type of head pain. Such head pain occuring in the absence of migraine is referred to as tension headache.

The sensory phenomena of classic headaches are believed to be due to intracranial vasoconstriction. The headache, on the other hand, is thought to result from dilation of the extracranial arteries and intracranial arteries (Adams, Feuerstein & Fowler, 1980). According to the theory put forth by Dalessio (1972), cranial vasodilation may be an organismic reaction to the vasoconstriction in which the body attempts to restore circulatory homeostasis in the cranium. Vascular distention in combination with a sterile inflammation brought about by the release of vasoactive substances at the site of the artery are the probable causes of the migraine pain which is felt (Dalessio, 1978).

Psychosomatic medicine has implicated stress as a factor in the pathological dysfunction underlying migraine. For example, Henryk-Gutt and Rees (1972) presented data where over one-half of 120 migraine attacks recorded during a two month period of observation were related in time to a stressful event. In addition the authors found that in approximately one-half of the 100 adults sampled with

migraine, the first incidence of this pain occurred in a period of emotional stress.

The notion that anxiety or stress may be important factors in the occurrence of migraine is not without a biochemical rationale regarding the mechanisms involved. Stress has been found to result in increases in the amount of epinephrine circulating in the blood. It has been suggested that increased epinephrine, in turn, brings about heightened platelet aggregation (Ardlie, Glew, & Schwartz, 1966; Langley, 1971). Recently hyperaggregability of platelets in migraineurs has been implicated in the release of serotonin which is a vasoactive substance (Dalessio, 1978; Deshmukh & Meyer, 1977). It has been suggested that a release of serotonin results in the vasoconstriction of large cranial arteries associated with the premigraine prodrome. With the subsequent depletion of the serotonin level the normal tonus of the arteries is lost and passive distention of the arterial walls takes place (Adams et al., 1980). Migraine pain is associated with a fall in serotonin plasma levels in over 85 percent of patients (Anthony & Lance, 1975). There is also a reduced pain threshold in the receptors of the arterial walls due to the permeation of a substance called plasmakinin which is synthesized in conjunction with serotonin (Fanchamps, 1974).

Heightened arousal prior to the migraine sequence would be expected from the above analysis . Dalessio (1972) observed that the evening before a migraine headache is

often characterized by excessive talkativeness and high spirits, unwillingness to retire, and increased appetite for food.

The theory that migraine pain may follow a period of stress and is felt as blood vessels dilate from the activity of vasoactive substances, suggests that the reduction of stress should reduce or perhaps eliminate the migraine attacks. As used here stress may refer to the environmental situations which produce sympathetic arousal in an individual, it may refer to the perceptions and cognitions by which the individual interprets the environment as being threatening, and stress might also be regarded as the psychophysiological reactivity of the individual to the environmental and cognitive events that have occurred.

The physiological mechanism thus far described accounts for reduced migraine via reduced stress at the precipitation or vasoconstriction stage of the sequence. However, this mechanism does not account for the alleviation of migraine once the pain has begun to occur. One would infer that biofeedback assisted hand warming, for example, at this point would only augment the vasodilation which is actually causing the pain. Yet, in some instances hand warming has been shown to be useful in aborting a headache after its onset (Sargent, Green & Walters, 1972; 1973). Sicuteri (1972) has offered one plausible explanation for this effect with the proposal that the hand warming procedure may influence the lowered pain threshold as well as the

humorally mediated inflammation of the vessel wall. Recent evidence of the efficacy of relaxation in producing greater pain tolerance (Hackett & Horan, 1980) lends support to Sicuteri's hypothesis.

In addition to biochemical aspects of stress however, one must bear in mind the effects of the autonomic nervous system when stressed. Activation of the sympathetic branch of the autonomic nervous system by a stressor results in pupil dilation, inhibition of salivation, secretion of sweat, constriction of blood vessels in the periphery of the body (causing cold hands and feet), dilation of blood vessels in the muscles, increased heart rate, increased blood pressure, and inhibition of digestive processes (Grings and Dawson, 1978). Several of these factors would aggravate the amount of pain produced when cranial arteries dilate from depletion of serotonin in the blood.

C. Temperature Training Biofeedback

Biofeedback of the moment-to-moment changes in hand temperature is a freqently utilized procedure for the treatment of migraine headache. Typically visual or auditory feedback indicating absolute temperature or temperature changes at the surface of the middle finger is provided to the subject who is instructed to relax and warm his or her hands. Temperature measured in this manner is directly related to the amount of blood flow in the finger (Surwit, Shapiro & Feld, 1976). As the biofeedback trainee relaxes

digit temperature increases to a maximum usually of about 96 degrees fahrenheit. Stress, on the other hand, results in a shunting of blood away from the extremities to the large muscles of the body. This has been characterized as a component of the "fight" or "flight" mechanism by which an organism prepares itself for vigorous physical activity.

The use of temperature training biofeedback in the treatment of migraine headaches was first reported by Sargent, Green, and Walters (1972, 1973) and Sargent, Walters, and Green (1973). The authors conceived of the idea to teach patients to warm their hands for the reduction of migraines after one of their subjects who was attempting to increase blood flow in the hands via EEG and EMG biofeedback mentioned spontaneous recovery from a migraine headache. The disappearance of the headache coincided with an abrupt 10 degree fahrenheit rise in differential hand and forehead temperature.

Almost a decade later there is as yet a controversy regarding the physiological mechanisms underlying biofeedback treatment effects for migraine. As was noted above, the migraine headache has been described as vascular pain caused by the exaggerated vasodilation of the extracranial arteries. Excessive vasodilation apparently occurs subsequent to stress induced vasoconstriction. Thus the migraine typically occurs some time after the stressful period. The logic for training in hand warming is based on the propositions that subjects who can voluntarily warm

their hands as part of a general relaxation response will be able to do so when they become stressed, or, will be able to do so instead of becoming stressed. In behavioural terms cold hands become a cue for stress and the need to practice relaxation.

It is important to note that the vasomotor activity of the blood supply network in the hands and the head may not operate in unison during a migraine. Serotonin has differential effects with respect to dilation and constriction depending upon the quantity of the substance, preexisting neurogenic tone, area of the body, and size of the blood vessel. Generally serotonin serves to constrict large arteries and veins, and dilate smaller vessels such as arterioles and capillaries (Anthony & Lance, 1975).

Price and Tursky (1976) have demonstrated high positive correlations between digital and extracranial blood volume changes for normal subjects as well as for migraine sufferers during nonmigraine periods. Whether or not such correlations exist while persons are suffering from migraine headaches is undetermined.

Price (1979) has concluded that research in the treatment of migraine by biofeedback is as yet equivocal. Although there are numerous studies which have reported a therapeutic benefit, methodological weaknesses in the research prohibit any clear cut confirmations. No controlled group outcome studies have been reported that show the superiority of digit temperature biofeedback over other

biofeedback procedures such as EMG training (Lake et al., 1979).The possibility exists that biofeedback per se plays a negligible role in this effect and that actually relaxation is the active ingredient in producing peripheral vasodilation (Price & Tursky, 1976).

Several studies have been published which attest to the efficacy of the treatment of migraine via biofeedback-assisted hand warming. These studies may be categorized under the headings of (a) anecdotal case reports, (b) systematic case studies, and (c) controlled group outcome studies.

**Anecdotal Case Reports**

The earliest publications of temperature biofeedback for migraines came out of the Menninger Clinic in the early 1970's (Sargent, Green, & Walters, 1972, 1973; Sargent, Walters, & Green, 1973). In these reports a treatment procedure was described in which a combination of autogenic training, passive relaxation, and biofeedback of the difference in temperature between hand and forehead was employed. Patients were trained to raise their hand temperature relative to the temperature of their forehead. The investigators' claim for clinical improvement of many of the patients studied was influential in prompting further research in the area. These studies themselves lacked adequate baseline data of headache intensity and statistical analyses of treatment effects were not conducted.

Turin (1975) treated seven migraine patients with hand warming alone in the absence of autogenic training. Headache improvement was reported for all seven patients but good headache frequency data was not collected. Other similar reports suggesting the efficacy of digit temperature biofeedback based on clinical trials without systematic recording of headache activity under both baseline and posttreatment conditions are those of Adler and Adler (1976), Kentsmith, Strider, Copenhaver, and Jacques (1976), Mitch, McGrady, and Iannone (1976), Peper (1973), and Weinstock (1972).

**Systematic Case Studies**

Wickramasekera (1973) used the differential (digit to head) temperature biofeedback procedure to successfully treat two patients with migraine. Headache activity was systematically recorded under both baseline and post-treatment conditions for temperature training. Frontal EMG biofeedback had initially failed to alleviate migraines in these individuals who had suffered with their headaches for more than 10 years. Medication and psychotherapy had also been tried in their past histories. Remarkably, temperature biofeedback proved effective within four sessions with the feedback being conducted on a once a week basis. In this time both patients learned to increase hand temperature four to five degrees. For one subject headaches were essentially absent at the end of three months while in the other quite low levels were maintained. Analgesic

medication was markedly reduced for both patients. An additional noteworthy aspect of this study is the fact that autogenic relaxation was not employed as a training strategy for temperature biofeedback.

Johnson and Turin (1975; Turin & Johnson, 1976) used an A-B-C design to measure the effects of bidirectional control over temperature in the hand only. Their work with three subjects indicated that frequency and duration of headaches actually increased over baseline levels during the hand cooling phase. Subsequent training in increasing digit temperature reduced the patients' migraines. Again here autogenic training was not employed in digit warming.

Research by Medina, Diamond, and Franklin (1976) obtained successful results in the reduction of migraine using a combination of frontal EMG biofeedback and digit temperature biofeedback. In this study 13 patients suffered from migraine and 14 patients had a combined migraine and muscle contraction symtomatology. Of those suffering from migraine alone 64 percent were reported improved while 30 percent of the mixed headache group were helped.

**Controlled Group Outcome Studies**

Andreychuk and Skriver (1975) reported a study in which feedback for hand temperature, combined with autogenic relaxation instructions proved to be no more effective than EEG alpha production combined with relaxation instructions, nor an autohypnosis treatment package which included instructions for relaxation. All patients were said to have

improved although insufficient information was provided regarding the criteria for improvement. Furthermore, no follow-up data were reported. An interesting aspect of this study was that subjects were pretested for hypnotic susceptibility and it was reported that highly susceptible subjects improved more than low susceptible subjects.

One study by Mullinex, Norton, Hack, and Fishman (1978) compared the effects of false feedback for hand temperature to that of veridical feedback. The results were that the majority of patients treated in each group showed improvements in migraine headache and there was no relationship between success in learning to raise skin temperature and a decrease in headache symptomatology.

Blanchard, Theobold, Williamson, Silver, and Brown (1978) compared two migraine treatment groups with a no-treatment control. One treatment consisted of feedback for fingertip temperature combined with autogenic training while the other group received Jacobsonian relaxation alone. Both treatment groups demonstrated greater improvements than controls. In addition, the Jacobsonian treatment group improved more than the autogenic feedback group initially, but no significant differences were found at a three month follow-up. The majority of patients in the two active treatment groups were improved. In a one year follow-up (Silver, Blanchard, Williamson, Theobald, & Brown, 1979) it was found that gains were maintained with no differential effects evident between the two treatment groups.

A recently published study of migraine treatment by Lake et al. (1979) provided a comprehensive analysis of the biofeedback response itself and its relation to migraine reduction. Treatment groups consisted of: (a) frontal EMG biofeedback, (b) digit temperature biofeedback, and (c) digit temperature biofeedback plus Rational Emotive Therapy (RET). A waiting list control group that self-monitored headache activity was also included. A reversal design within each treatment session was used to assess the extent of bidirectional control over the target physiological response. Treatments consisted of eight to ten, 30 minute sessions, scheduled twice per week. Results indicated that digit temperature feedback alone and in conjunction with RET did not prove to be significantly more effective than controls. All six of the EMG subjects reduced headache activity to two-thirds or less of the baseline level in a three month follow-up. However, only four of the digit temperature biofeedback subjects improved by the criterion of 33 percent reduction and only two of ten digit temperature plus RET subjects so improved. Statistical analysis indicated only the EMG treatment to be superior to self-monitoring.

Experimental analyses of bidirectional control in biofeedback training indicated that digit temperature performance was not maintained over time and was unrelated to improvement in headache activity. Evidence for bidirectional control of the digit temperature response was

found in only one third of the digit temperature sessions. EMG subjects met a performance criterion for bidirectional control on 85 percent of the sessions and self-reports indicated that EMG biofeedback performance was an easier task to learn.

Lake et al. (1979) noted that the poor bidirectional control results for digit temperature are consistent with findings reported in other well controlled group outcome studies using both normal subjects and migraine patients (Packer & Selekman, Note 1; Price & Tursky, 1976; Surwit, Shapiro, & Feld, 1976). Lake et al. have argued that adequate within subjects experimental designs are necessary to demonstrate response control because temperature rise with or without feedback is common during baseline conditions. However, one might argue that practice in bidirectional control involves undesireable practice of a stress response. Valuable time for practice in relaxation is lost to this practice of cooling hands and tensing muscles. Furthermore, nonfeedback conditions which existed during the pre-training baseline phase of each session deprives the subject of potentially valuable information while his or her body is relaxing to the degree which the subject can already produce.

The results of Lake's et al. (1979) research indicated that temperature control is not readily achieved through eight biweekly sessions of bidirectional control training. Furthermore, when bidirectional control is achieved it does

not necessarily mean that headaches will reduce by noncontingent daily home practice of nonfeedback assisted "relaxation" as was applied. Perhaps a more thorough approach would obtain better results by providing more practice in biofeedback assisted hand warming as well as further procedures for applying and generalizing the skills learned.

### D. Biofeedback, Relaxation and Response Stereotypy

The critical components of biofeedback treatment procedures for the reduction of migraine have not as yet been determined. Autogenic relaxation training is an integral part of the migraine treatment packages used at the Mayo Clinic (Fahrion, 1978) and the Menninger Foundation (Sargent, Green & Walters, 1972; 1973). Autogenic training itself is a method of psychosomatic self-regulation which is used to bring about the gradual acquisition of autonomic control (Schultz & Luthe, 1969). This autonomic control is developed through a strategy of passive concentration by which the individual progresses toward a state of relaxation while maintaining a detached attitude toward his actual progress. As in hypnosis the person often focuses his attention on visual, somatic and auditory imagery involving such effects as hand warmth or muscle relaxation in order to induce these physiological changes. When biofeedback training is used in combination with autogenic relaxation it is seen as a facilitator of psychosomatic response control.

At present the efficacy of biofeedback procedures alone in the treatment of migraine is unclear. Only one controlled group outcome study has been conducted where biofeedback in the absence of additional relaxation training was employed (Lake et al., 1979). However, temperature training alone has been effectively used with migraine subjects in several case studies (Johnson & Turin, 1975; Turin, Note 2; Turin & Johnson, 1976; Wickramasekera, 1973).

The two most common forms of biofeedback treatment for headaches are electromyographic (EMG) and temperature training. Consideration of the training format involved in each of these types of biofeedback indicates a great deal of similarity to the training format employed in autogenic training. In each case the subject is instructed to sit quietly and assume an attitude of passive concentration. In order to gain control of the bodily function being monitored with biofeedback the subject is told not to think about muscle tension or body temperature but instead to direct his or her attention to the feedback signal and to passively allow it to change in the desired direction (Hiebert, Note 3). Actively striving to hand warm or decrease muscle tension frequently produces an opposite result to that desired (Karlins & Andrews, 1972; Libo & Fehmi, Note 4).

Autogenic relaxation and biofeedback training also appear to have aspects of decreased arousal in common. In biofeedback learning however one might say that control over a specific physiological response is the primary goal and

general arousal reduction is a secondary outcome of the training procedure. For example Yates (1980, pg. 1) has recently defined biofeedback as "the display of some aspect of the physiological functioning of the individual with the expectation that observation of the characteristics of the display will enable the individual to attain increased voluntary control over the physiological function being displayed." Alternatively, general decreased arousal is the main goal of autogenic training and subsequently particular desireable physiological changes are enhanced. Decreased arousal may include lowered levels of muscle tension, skin conductance, respiration rate, heart rate, and blood pressure, as well as pupilary constriction and peripheral vasodilation (Budzynski, 1973; Germana, 1974). The main difference between any form of biofeedback learning and autogenic training is in the specificity of physiological control which is afforded and monitored by the biofeedback learning. In autogenic training alone reduced levels of arousal in particular physiological systems can only be assumed.

EMG biofeedback training like autogenic training may be viewed as an indirect means of producing the parasympathetic vasodilation that is sought in migraine treatment via temperature biofeedback. With EMG biofeedback an attempt is made to reduce muscle tension throughout the body. Reduced muscle tension is believed to promote a shift from sympathetic to parasympathetic dominance in the autonomic

nervous system (Gellhorn, 1967).

The relationship between skeletal musculature functioning and the autonomic nervous system has been delineated by the work of Gellhorn and Kiely (1972). These authors distinguish between two systems termed ergotropic and trophotropic. The ergotropic system is responsible for the syndrome of increased sympathetic discharges, elevated muscle tone and cortical excitation. The trophotropic system is responsible for increased parasympathetic discharges, decreased skeletal muscle tension and reduced cortical excitation. According to Gellhorn and Kiely's model the interplay between ergotropic and trophotropic systems may be altered by two distinctly different operations: (a) through direct stimulation of ergotropic or trophotropic cerebral centers such as the hypothalamus; (b) through indirect alteration of the activity of these two systems. With regard to the latter, reduced muscle tension results in reduced afferent input impinging on the reticular formation and hypothalamus. This in turn causes a decreased rate of hypothalamic-cortical discharge and a dominance of the trophotropic system through the mechanism of reciprocal innervation.

On the basis of Gellhorn and Kiely's model one would predict that increased parasympathetic responding would be enhanced by any form of muscle relaxation treatment such as autogenic training, progressive relaxation, or EMG biofeedback. A critical question is whether relaxation

training will provide sufficient parasympathetic effects to inhibit the mechanisms that produce the exaggerated vasodilation which migraine sufferers are prone to experience. Lance (1973) has shown that migraine patients, even in periods of remission, have more variable and larger pulsations of the superficial temporal artery. For this reason he has characterized migraines in terms of vascular instability. Tunis and Wolff (1953) also found greater variability in the temporal artery pulse of migraine subjects than normal. They demonstrated that the amplitude of pulsations of the temporal arteries increased at the onset of migraine headaches. However, increased vasoconstriction was found during the pre-headache period when scotomas were present.

Stoyva (1977) has written about vascular instability in migraine patients as a form of response stereotypy that has become pathological due to frequent triggering of the stress response. He hypothesized that frequent triggering of the vascular response results in a loss of adequate homeostatic control over vascular changes. Selby and Lance (1960) have suggested that the variability of vasomotor functioning in the cranial arteries of migraine patients may reflect more of a general instability of the autonomic nervous system than an independent pathophysiology in the central vasomotor control system. Whether or not this is true it is known that the autonomic nervous system does tend to respond as a whole when stressed (Lacey, Bateman, & Van Lehn, 1953). This fact

supports the inclusion of general relaxation in biofeedback training formats for migraine headache.

In view of what has been said above about the indirect alteration of the activity of ergotropic and trophotropic systems it seems plausible that for some persons migraine headaches may be produced indirectly due to the effects of high levels of muscle tension. In other words migraine headaches may be caused by various patterns of stress responsiveness in the vasomotor and skeletal musculature systems. Hypothetically any pattern of stress may eventually trigger the vascular mechanisms which lead to the migraine. In this light it is interesting to note that chronic high levels of tension in the muscles of the head and neck are common among migraine sufferers (Pozniak-Patewicz, 1976). Furthermore, Bakal and Kaganov (1977), have presented evidence that migraine patients as a group display higher frontal EMG activity than muscle contraction headache patients and headache-free controls. These authors concluded that patients diagnosed with muscle contraction headache and patients diagnosed with migraine appear to have a similar physiologic predisposition for headaches. Their treatment results also supported this notion of similar predisposition as they found that frontal EMG biofeedback training was equally effective for migraine and tension headache subjects. Bakal and Kaganov (1977) also examined the pain locations reported by migraine and muscle contraction headache patients and found considerable similarities

between the two groups. The similarities led the authors to question the utility of the diagnostic system recommended by the Ad Hoc Committee on Classification of Headache.

The fact that there is variability in the stress responsiveness of different individuals has led biofeedback practioners to develop a technique for measuring a person's physiological stress profile (Stoyva, 1979). While monitoring various modes of physiological activity (eg. muscle tension, hand temperature) the individual is subjected to mental stressors such as performing mental arithmetic or imagining an upsetting event which the person has experienced. In this manner idiosyncratic patterns of stress are determined in terms of changes in the various levels of physiological activity. Stoyva (1979) described the stress profile procedure which he uses as consisting of 14 minutes of relaxation, six minutes of stress (subtracting serial seven's) and six minutes of recovery, during which the client attempts to relax after having performed the mental arithmetic. Variants of this approach are described by Fair (1979) and Stroebel (1978). All three of these authors mention the use of the stress profile in the context of treatment for anxiety. Typically, a clinical judgement is made of stress profile results and biofeedback is provided in the physiological modality which the therapist feels to be most "aberrant." The present author is unaware of any reports where the stress profile has been used to make treatment decisions in the context of migraine headache.

To date, no clear cut, quantifiable methods have been described for interpreting a stress profile. Stoyva (1978) mentions that some clients may show a favored physiological response, such as muscle tension, or cool hands in all three phases of the stress profile testing. Other clients demonstrate usual levels of relaxation initially but then become excessively aroused in one or more physiological systems during the stress task. Still others indicate a problem in failing to recover from increased arousal brought on by the stress.

The issue of physiological response stereotypy in migraine headache has been investigated by Cohen, Rickles, and McArthur (1978). The term stereotypy is used to denote a reproducible pattern of physiological changes in an individual's reaction to a variety of stressors (cf. Lacey & Lacey, 1958; Roessler, Greenfield & Alexander; 1964). Cohen et al. hypothesized that migraine patients would exhibit a more stereotypic physiological response profile across various forms of stress than would a group of headache free subjects. The stressors used included orienting to a tone, time estimation, reaction time and mental arithmetic. The physiological measures taken were head and hand temperatures, frontal EMG, heart rate, skin conductance level, and digital pulse amplitude. Greater physiological stereotypy in migraine subjects was evidenced by a more stable pattern in the amounts of physiological activity across the tasks than that shown by the control group.

The concept of response stereotypy is important to the rationale for treating migraines via biofeedback. If the pattern of physiological reactivity varied considerably among different forms of psychological stress then biofeedback training with any one physiological system would only be expected to have capricious effects on an individual's physiological functioning.

The area of physiological response patterning has been reviewed by Hiebert (Note 5). This author cites considerable support for the notion of response stereotypy as well as several studies which have demonstrated evidence of response specificity. Response specificity is the elicitation of a particular response pattern across individuals using a specific stressor. Response stereotypy and response specificity at first glance appear to contradict one another, but as Hiebert (1980) has pointed out it is conceivable that factors such as experimental procedure, physiological variables measured, and data analysis procedures may determine whether a specific or a stereotypic response is demonstrated.

Another important aspect of response stereotypy is the fact that stereotypy refers to consistency in the physiological reactivity of one individual. Stereotypy does not mean that several persons will display the same pattern of physiological arousal. Rather it refers to the finding that a person's idiosyncratic reaction to stress is enduring. For example, Lacey and Lacey (1962) found that

stereotypic reactivity endured throughout four years in longitudinal follow-up.

This author has been unable to find any convincing research that shows that migraine suffers as a group display a common form of physiological reactivity to brief presentations of stressors. Research by Cohen et al. (1978) demonstrated that migrainers and nonmigrainers exhibited similar average changes in head and hand temperatures across several psychological stressors.

Some studies have obtained differences between migraines and nonmigraines in tonic reflex vasodilation of the hands to a heat stimulus (Appenzeller, 1969; Appenzeller, Davison & Marshall, 1968; Downey & Frewin, 1972; Elliot, Frewin & Downey, 1973). However, these results were not obtained in the research of French, Lassers and Desai (1967) nor in the research of Hockaday, Macmillin and Whitty (1967). In his review of the literature pertaining to generalized vasomotor dysfunction in migraine, Morley (1977) concluded that all the studies showing any differences in tonic reflex vasodilation had serious methodological flaws. He argued that there is no evidence to date supporting the notion of generalized abnormal vasomotor control among migraine patients.

Price and Tursky (1976) found the learning rates of migraine sufferers to be markedly different from normal subjects in a one-session attempt at training autonomic relaxation (peripheral vasodilation). A number of training

procedures in addition to temperature biofeedback were compared and it was found that normal subjects did learn to increase digital blood volume over time, whereas migraine sufferers remained the same or decreased blood volume. The authors interpret their results to suggest that the autonomic system plays an important role in the development of migraine.

Hypothetically one might expect that patients with some particular somatic complaint such as migraine would be particularly susceptible to elevated stress-induced activation of the physiological system associated with the somatic problem. However such elevated stress-induced activation may not occur as a result of the brief presentations of stress typically used in the laboratory. Indeed, there have been no reports of migraine being elicited subsequent to laboratory stress testing. Possibly, the sequence of cranial arterial vasoconstriction and rebound vasodilation that migraine sufferers demonstrate requires prolonged or excessive stressing of what are otherwise normally functioning stress reactive physiological systems.

Equivocal results in the reduction of migraine among various forms of treatment such as EMG biofeedback, temperature training biofeedback, and various forms of relaxation training may be due to the lack of consideration for individual stress profiles. Hypothetically, positive results which have been obtained by various treatments in

the past may have been due to the fact that the particular treatment modes used accidentally corresponded to the psychophysiological systems that were most responsive for some of the individuals included. A more practical approach may be to match treatment modes according to the reactive response systems which the patients display.

**E. Summary**

Psychophysiological stress has been implicated as a factor in the pathological dysfunction underlying migraine headaches. Migraine pain is considered to be caused primarily by excessive dilation of cranial arteries subsequent to a period of heightened arousal or stress. However, evidence has been provided that patients diagnosed with migraine and patients diagnosed with tension headaches have a similar physiologic predisposition for headaches in terms of cranial muscle tension.

Several forms of biofeedback therapy and relaxation training have proven effective in the treatment of the migraine and to date the comparisons among treatments are equivocal. One might conclude that some forms of treatment are more effective for some types of patient. It is known that patterns of physiological stress in the various body systems can differ among individuals. For this reason some clinicians working with patients who are anxious have speculated that biofeedback for any individual may be more effective when the feedback modality matches the

physiological system which is most reactive. This rationale might also apply to migraine sufferers since it has not been determined that as a group they tend to become physiologically stressed in any particular manner. Perhaps treatment for the migraine patient would be more effective if patients were trained how to recognize tension and how to relax, guided by biofeedback in the modality which for them was most responsive.

## F. Research Questions

The present investigation was conducted to answer the following questions:

1. Is digit temperature biofeedback in combination with EMG biofeedback more effective in the management of migraine than either digit temperature biofeedback alone, or EMG biofeedback alone?

2. Do EMG reactive subjects in EMG treatment and temperature reactive subjects in temperature treatment experience a greater reduction in migraines, than subjects with relatively low EMG reactivity who are given EMG treatment and subjects with relatively low temperature reactivity who are given temperature biofeedback treatment?

3. Are differential biofeedback treatment effects more apparent when subjects are grouped according to criteria based on amount of physiological reactivity or when they are grouped according to duration of physiological reactivity?

4. What are some of the differential EMG and temperature performance characteristics among the various biofeedback and stress reactivity groupings of subjects?

## III. METHOD

### A. Subjects

Subjects were obtained through a press release issued by the University Public Relations Department to all media in the Edmonton vicinity. From those who responded to the advertisement 60 subjects were selected for inclusion in the study according to criteria set from the considerations discussed in Adams et al. (1980), and Blanchard et al. (1979). There criteria were as follows:

1. Subject's age falls between 18 and 55 years inclusively.
2. Subject's headaches have occurred one or more times per month over the past 2 years.
3. Female subjects are not currently using oral contraceptives.
4. Subject is not currently receiving any form of psychotherapy.
5. Subject does not suffer from a convulsive disorder.
6. Subject does not have any form of heart disease or disorder.
7. Subject must report YES to three out of the following five items,
   a. Does the head pain sometimes exist on one side of the head only?
   b. Is the head pain generally pulsative (or throbbing)?
   c. Does nausea or vomiting generally accompany the headache?

d. Does sensitivity to light generally accompany the headache?

e. Has the headache already been diagnosed as a migraine by some physician?

Each subject who agreed to participate in the study was required to sign a treatment contract with the experimenter in which the responsibilities of subject and experimenter were clearly stated (see Appendix A). In addition, all subjects were required to provide proof of having received a recent medical examination and to have their physician sign a statement that there is no medical reason why they should not participate in the study (see Appendix B). Five subjects were dropped from the study for failing to keep regular headache data records and/or poor attendance at treatment sessions. Table 1 (see next page) indicates the characteristics of the study population which was obtained from a questionnaire.

Table 1

Characteristics of Study Population (N=55)

| | | Number Answered True |
|---|---|---|
| 1. | Able to tell that a migraine is coming before the headache actually begins. | 40 |
| 2. | Able to tell that a migraine is coming through visual changes or distortions. | 26 |
| 3. | Able to tell that a migraine is coming by some other sensory or motoric change. | 31 |
| 4. | The head pain frequently exists on one side of the head only. | 48 |
| 5. | The head pain usually exists in the temporal regions (at eye level on the side of the head). | 41 |
| 6. | The head pain usually exists in the forehead region, between the eyebrows and hairline. | 19 |
| 7. | The head pain usually begins in the neck at the base of the head and then radiates toward the temporal and forehead regions. | 25 |
| 8. | The headaches occur in many different regions from time to time. | 23 |
| 9. | The head pain usually occurs in the region at the top of the head. | 6 |
| 10. | Frequently the headaches are throbbing, pulsating headaches. | 49 |
| 11. | The headaches are usually characterized by pressure on the head, the sensation of which might be described as a tight band across the forehead and around the head. | 35 |
| 12. | The headaches usually only occur during menses. | 3 |
| 13. | Nausea or vomiting generally accompany the headaches. | 42 |
| 14. | Sensitivity to light generally accompanies the headaches. | 50 |
| 15. | Sensitivity to sound generally accompanies the headaches. | 47 |
| 16. | Tears and nasal stuffiness generally accompany the headaches. | 24 |

Table 1 (cont'd)

| | Number Answered True |
|---|---|
| 17. Your headache has been diagnosed as a migraine by a physician. | 54 |

Number of females = 44 Number of males = 11

Mean age = 38.7 years Age Range = 20 - 54 years

Mean number of years with migraine headaches = 21

| Reactivity Groups | Treatment | Mean Age | Age Range | Males | Females |
|---|---|---|---|---|---|
| EMG | EMG | 39.1 | 30-50 | 1 | 8 |
| | Temperature | 34.5 | 24-45 | 1 | 7 |
| | Combined | 42.1 | 30-52 | 2 | 8 |
| Temperature | EMG | 40.2 | 20-53 | 3 | 7 |
| | Temperature | 35.0 | 22-53 | 1 | 7 |
| | Combined | 39.8 | 29-54 | 3 | 7 |

| Responsivity Groups | Treatment | Mean Age | Age Range | Males | Females |
|---|---|---|---|---|---|
| EMG | EMG | 38.6 | 30-50 | 1 | 8 |
| | Temperature | 35.8 | 24-45 | 0 | 9 |
| | Combined | 41.6 | 30-52 | 3 | 7 |
| Temperature | EMG | 40.6 | 20-53 | 3 | 7 |
| | Temperature | 33.4 | 22-53 | 2 | 5 |
| | Combined | 40.3 | 29-54 | 2 | 8 |

## B. Research Design

### Part 1

The research design was essentially a three (treatments) by two (groups) repeated measures across treatment phases format. The eight-session program for each of the three treatment groups was carried out in two segments with half (30) of the subjects beginning treatment in June and the other half beginning treatment in July. A general introduction and contract signing session was held in mid-May. Subjects were asked to monitor headaches from this point on.

During the initial four weeks of baseline in May, all 60 subjects were seen for a psychophysiological stress profile session. On the basis of this profile subjects were divided into two main groups by a median split procedure. The 30 subjects whose stress profiles demonstrated relatively high EMG reactivity levels with relatively low temperature reactivity formed one group; the 30 subjects whose stress profiles indicated relatively high temperature reactivity with relatively low EMG reactivity formed the second group. The subjects from each of these two groups were then randomly assigned to the three treatment conditions.

The initial treatment by groups arrangement may be illustrated as follows:

| GROUPS | TREATMENTS | | |
|---|---|---|---|
| | EMG | Temperature | Combined |
| EMG Reactive | n=9 | n=8 | n=10 |
| Temperature Reactive | n=10 | n=8 | n=10 |

Headache intensity was evaluated over three four-week phases of the experiment including baseline, treatment, and follow-up.

**Part 2**

A second analysis was performed on the treatment data subsequent to the three way repeated measures analysis described in Part 1. In Part 1 subjects were classified as EMG or temperature reactive according to criteria based on the level increase in EMG and the level decrease in temperature during the stress activity. An alternative way of considering stress reactivity is in terms of the amount of time it takes for a person's physiology to recover from the stress event. In order to compare results obtained when subjects are categorized according to level of reactivity with results obtained by categorizing them according to duration of reactivity the second analysis was conducted. For the sake of clarity, duration of reactivity will herein be referred to as responsivity.

In Part 2 subjects were reclassified by the median split procedure into two groups. One half of the subjects whose stress profiles demonstrated relatively long periods of EMG responsivity with relatively short temperature responsivity formed one group; the remaining half of the subjects who demonstrated relatively long periods of temperature responsivity with relatively short periods of EMG responsivity formed the second group. The grouping of subjects by the recovery calculations resulted in a reclassification of 11 out of the 55 subjects. The number of subjects in each of the treatments by group cell combinations was as follows:

| GROUPS | TREATMENTS | | |
|---|---|---|---|
| | EMG | Temperature | Combined |
| EMG Responsive | n=9 | n=9 | n=10 |
| Temperature Responsive | n=10 | n=7 | n=10 |

## C. Apparatus and Facilities

Biofeedback sessions were carried out in a 20′ x 30′ laboratory. Subjects were seated in a comfortable lounge chair adjacent to a table containing the biofeedback equipment.

EMG levels were measured using an Autogenics Systems Incorporated 1700 electromyograph. The auditory and digital feedback levels of the EMG were generated using a one second response averaging mode. A 100-200 Hz frequency bandpass was used as recommended by the manufacturer for general muscle relaxation training. Also, frontal electrode placement and impedence levels of less than 10,000 ohms were used as recommended by the manufacturer. Silver-silver chloride type electrodes were used for all subjects. Biofeedback was provided to the subjects via variable frequency auditory clicking and a meter gauge which visually displayed EMG levels in microvolts.

Temperature levels were measured using an Autogenics Systems Incorporated 2000 temperature monitor. Research grade thermisters were attached by tape to the middle phalanx of the middle finger of the nondominant hand (Surwit, et al., 1976). Temperature level feedback was provided to the subject via a variable tone and a meter gauge indicating fahrenheit degrees. Temperature and EMG levels were processed simultaneously using an Autogenics Systems Incorporated 5600 data acquisition centre and printer assembly. This assembly was calibrated to provide a microvolt/second integrated voltage value. The average level of response over the final 10 seconds of each minute was recorded by the printer unit during treatment sessions.

## D. Stress Profile Procedures

Physiological stress profiles (see Appendix C) were obtained by a modified version of the general guidelines described by Stoyva (1979). First the subject was instructed to relax with his or her eyes open for 15 minutes while EMG and temperature levels were being monitored. After 15 minutes the subject was instructed to close his eyes and to continue relaxation. Following three minutes of relaxation with the eyes closed a three minute period of stressing was conducted in which the subject was instructed to serially subtract seven from 1,000 as fast as possible until told to stop. This was performed silently and with the eyes closed. At the end of three minutes the therapist asked the subject to indicate his answer. Finally, a five minute recovery period was conducted during which the subject was instructed to keep his eyes closed and again relax. Feedback was not provided during stress profile sessions.

### Differential Reactivity Calculations

Instantaneous EMG and temperature levels were monitored every 20 seconds. EMG reactivity was calculated by subtracting the mean EMG level during the final two minutes of eyes-closed relaxation prior to stressing, from the mean EMG level obtained during the three minutes of mental arithmetic. Negative reactivity scores that were obtained were considered to indicate no reactivity and therefore assigned the value of zero. EMG artifact that may have been caused by swallowing or moving the head was defined as any

EMG reading that was at least seven microvolts higher than the previous or subsequent reading. Such artifact was not included in the computations of means.

Temperature reactivity was calculated somewhat differently than EMG reactivity due to the nature of the temperature data which demonstrates a high degree of serial dependancy. Temperature reactivity was calculated by subtracting the lowest temperature value obtained during the three minute stress period from the initial temperature reading during the stress period. This value was then subtracted from the difference obtained when the final temperature score of the three minute eyes-closed relaxation period was subtracted from the initial temperature of this relaxation period. Thus the temperature reactivity value accounted for the slope of the temperature values that already existed prior to stressing. As was the case with EMG, negative temperature reactivity scores that were obtained were considered to indicate no reactivity was present. Therefore they were assigned the value of zero.

Temperature and EMG reactivity values were converted into t scores. The pair of temperature and EMG reactivity scores for each subject were plotted on a scattergraph and subjects were divided into two groups, a) relatively high EMG reactivity or b) relatively high temperature reactivity, by means of a median split procedure. These two groups constituted the groups used for analyses in the experimental research design.

E. Treatment Procedure

Subjects in all three treatment conditions recorded daily headache intensity and medication consumption during the three four-week phases of baseline, treatment and follow-up, using a grid and rating scale similar to that developed by Budzynski, Stoyva, Adler, and Mullaney (1973). On the cards provided the subject was asked to rate headache activity for each waking hour as follows: "0" = no headache; "1" = low level, only enters awareness when you think about it; "2" = aware of headache most of the time but it can be ignored at times; "3" = painful headaches but still able to continue job; "4" = severe headache, difficult to concentrate with undemanding tasks; "5" = intense, incapacitating headache. Data cards were collected at each training session.

Three graduate students with experience in counselling and biofeedback training served as therapists during treatment. A number of subjects from each treatment group were assigned to each therapist according to schedules of availability.

Group 1: EMG Biofeedback Training

All subjects received eight individual training sessions twice weekly over four weeks, plus one follow-up session held eight weeks after the final training session. The EMG treatment format and instructions to subjects (see Appendix D) were similar to those used by Hiebert (1979). Subjects received 20 minutes of EMG biofeedback training in

each session. Temperature was also monitored. Each session was preceded by a discussion of the rationale for treating migraines by biofeedback. The rationale included suggestions regarding observations, awareness, and strategies that may prove useful in training.

At the end of each session subjects were asked to spend five minutes writing down a description of the strategies which they found successful and to identify any feelings or sensations which appeared to be associated with slower clicking. They were also instructed to take home what they had written down and to practice their strategies 10-15 minutes twice daily in an attempt to duplicate outside the lab. the same feeling state associated with slow clicking biofeedback.

In sessions five, six, seven and eight subjects were asked to practice controlling muscle relaxation in the absence of the auditory feedback. This practice or "weaning" lasted for five minutes and took place after 15 minutes of training. At the end of the five minute practice the experimenter indicated how well the subject progressed without the auditory feedback. Subjects were also instructed to begin to use the cognitive strategies, which they had found successful at any time during the day when they were in a stressful situation.

**Group 2: Digit Temperature Biofeedback Training**

Laboratory training Procedures for Group 2 were identical to those used in Group 1 with the exception that

temperature biofeedback was provided while EMG was only being monitored (see Appendix E). In addition subjects were provided with "Biotic Band" digit temperature indicator units which they were instructed to use during one of the twice daily home practice periods (see Appendix F).

**Group 3: EMG and Digit Temperature Biofeedback Combined**

In this group subjects received EMG biofeedback in the first two sessions and temperature biofeedback in the next two sessions. Thereafter subjects simultaneously received visual feedback from the EMG and temperature biofeedback instruments. Auditory feedback was presented from the EMG biofeedback instrument alone. The last four treatment sessions followed the same format as that used in Groups 1 and 2. Basically the same instructions as those used in the other groups were given to the subjects in Group 3.

**Follow-Up**

An eight week follow-up session was conducted with the subjects from each of the six treatment-by-group combinations of groups. Daily headache monitoring sheets were collected from these subjects during this follow-up session.

## F. Dependent Measures

Hourly headache intensity ratings for each subject were averaged to obtain a mean headache rating per day for each period including baseline, treatment, and one-month follow-up. This score was computed as Hr/d, where Hr = sum

of headache ratings over the experimental period being considered, and, d = number of days in the experimental period. The headache intensity variable was selected for use because of its sensitivity to changes in headache duration and intensity (Blanchard, 1978).

## G. Ethical Considerations

All subjects used in this study were volunteers. Although each was required to meet a number of criteria regarding migraine symptomatology for inclusion in the study, no diagnosis of migraine or any other disorder was made by the researchers involved in the study. Each subject was required to see a medical doctor regarding the head pain before entering the treatment program. A medical consent form was provided for each of the subjects to obtain a physician's consent for participation in the biofeedback relaxation program. (See Appendix B).

Subjects were informed about the nature of the study at the outset and the experimenter agreed to provide a summary of the results obtained after the program was completed. A treatment contract outlining the responsibilities of client to therapist and therapist to client was signed by both parties before treatment began (See Appendix A). Subjects who wished to drop out of treatment were free to do so at any time.

## IV. RESULTS

Data analyses are organized for presentation under three different headings:

A) Treatment Effects and Psychophysiological Reactivity - where subject groups were formed on the basis of differential changes in EMG and temperature levels during stress profiles. Headache data are analysed among the three treatments and two reactivity groupings.

B) Treatment Effects and Psychophysiological Recovery - where subject groups were formed on the basis of differential rates of recovery in EMG and temperature levels during stress profiles. Headache data are analysed among the three treatments and two recovery groupings.

C) Biofeedback Training Data - where physiological learning curves among treatment groups are compared.

### A. Treatment Effects and Psychophysiological Reactivity

#### Stress Profile Data

Figure 1 is a scattergraph illustrating the dispersion of subjects according to their levels of EMG and temperature reactivity to stress. Each dot represents one subject. EMG and temperature reactivity values have been converted to T-scores with a mean of 50 and a standard deviation of 10. The diagonal line indicates the division of subjects into two groups: (a) *EMG Reactive* - relatively large EMG increases with relatively small temperature decreases, and (b) *temperature Reactive* - relatively large temperature

decreases with relatively small EMG increases.

Figure 2 illustrates the mean EMG values that were obtained during the stress profiles for the two reactivity groups. It can be seen from the graph that the group defined as EMG reactive had a considerably greater mean increase in EMG from relaxation to stressing than did the group defined as being temperature reactive. The mean EMG level during the three minute stress period was 1.326 microvolts higher than the mean EMG level during the preceding three minute relaxation period for the group classified as EMG reactive. However, the group classified as temperature reactive demonstrated little mean change in EMG during the mental stress phase, as their average EMG actually decreased by 0.039 microvolts. Welch's adjusted t test for unequal variance (Ferguson, 1971) indicated that these mean changes in EMG level from relaxation to stressing for the two reactivity groups were significantly different ($t(43) = 4.44$, $p < .001$).

Figure 3 illustrates the mean temperature values that were obtained in the course of the stress profiles for the two reactivity groups. Contrary to the EMG response which increases during stress, finger temperature tends to decrease when a subject is asked to perform mental arithmetic. On the graph it can be noted that the group which was classified as temperature reactive demonstrated a drop in temperature over the course of the three minute stress period. This temperature drop followed an upward

trend that can be observed during the previous three minutes of relaxation. The EMG reactive group on the other hand did not evidence much of a drop in temperature from the stress task, over and above the gradual decline which already existed during the previous relaxation time segment. A comparison of mean temperature reductions during stressing between the two reactivity groups indicated a statistically significant difference (Welch's $t(35) = -4.78$, $p < 0.001$). As described above, calculations of temperature drop during the stress period were adjusted to account for the trend in temperature which was evidenced over the previous three minutes of relaxation. On the basis of the adjusted score for temperature change it was found that the temperature reactive subjects showed a mean drop in finger temperature of 0.92 degrees fahrenheit. The group classified as EMG reactive demonstrated a mean drop of 0.12 degrees fahrenheit.

**Summary**

The median split procedure was successful in identifying two groups of subjects who demonstrated differential psychophysiological reactivity to stress as measured by frontal EMG and finger temperature. Mean comparisons of EMG changes and temperature changes for the two reactivity groups indicated that one group presented a profile of relatively high EMG reactivity with relatively low temperature reactivity; while the other group demonstrated a stress profile of relatively high temperature

reactivity with relatively low EMG reactivity.

**Daily Headache Record**

Headache data were subjected to two (groups) by three (treatments) by three (periods) repeated measures analyses of variance with subjects nested in treatments and groups. Mean daily headache levels were calculated over the three phases of the experiment. The research design is presented in Figure 4.

The Analysis of Variance indicated a significant period effect for this dependent measure: Conservative $F(1,42) = 13.29$, $p < 0.001$. The treatment group means are listed in Table 1 (see next page) and portrayed graphically in Figure 5. Summary tables for the Analyses of Variance appear in Appendix G. Seven subjects were excluded from this analysis in order to maintain an equal number of subjects in each treatments-by-groups cell combination. Random selection procedures were used to exclude each of the subjects from the required cells leaving a total number of 48 subjects.

No significant two-way or three-way interactions were obtained for the headache intensity variable. The significant period effect indicated that subjects obtained a reduction in migraines regardless of the stress profile grouping or type of biofeedback treatment employed. Scheffe post hoc comparisons were made to determine the significant phases of headache reduction among the three treatment periods. A significant reduction in headache was found from pretreatment to posttreatment and from treatment to

Table 2

Mean Headache Levels Across Reactivity Groups,

Treatments and Periods.

| Reactivity Group | Treatment Mode | Period | | |
|---|---|---|---|---|
| | | Pretreatment | Treatment | Posttreatment |
| | | $T_1$ | $T_2$ | $T_3$ |
| EMG Reactive | EMG | 4.8 | 6.3 | 3.6 |
| | Temperature | 8.3 | 5.1 | 3.6 |
| | Combined | 11.4 | 10.6 | 8.3 |
| Temperature Reactive | EMG | 9.9 | 8.8 | 6.3 |
| | Temperature | 8.3 | 9.0 | 6.1 |
| | Combined | 12.0 | 8.7 | 7.3 |
| | Period Means (N=48) | 9.1 | 8.0 | 5.9 |

posttreatment: *Sch* $F(2,162) = 1.488$, $p < .05$. Significant differences were not obtained from pretreatment to the treatment period.

Figure 5 illustrates considerable variability in pretreatment headache levels among the six treatments-by-groups combinations. It can be noted that this variability is quite reduced during the posttreatment period. The overall pattern of mean reductions in headache does not indicate any strong suggestive trends toward differential effectiveness among the treatment combinations.

The overall change in headache ratings among the three treatment groups from pre-treatment baseline to one-month follow-up indicated a 36 percent average reduction. This clinical improvement is even more impressive when looked at in terms of "severe" headaches. It was found that 32 of the 55 subjects included in the study obtained more than a 50 percent reduction in headaches rated as "4" (severe headache, difficult to concentrate with undemanding tasks) or "5" (intense, incapacitating headache).

**Summary**

The overall pattern of reductions in means from pretreatment to posttreatment failed to support the results which had been predicted. There was no evidence that treatment effects were enhanced when the treatment modality matched the more reactive physiological system. A statistically significant main effect was obtained for the period factor. Post hoc analyses indicated a significant

overall reduction in headache from pretreatment to posttreatment and from treatment to posttreatment.

## B. Treatment Effects and Psychophysiological Recovery

### Stress Profile Reclassification

The analyses of headache change presented thus far has been based on the differentiation of subjects into stress reactivity groups. Reactivity among subjects was determined by the extent of change in EMG and temperature levels from a period of relaxation to a period of stress. Another hypothetically useful way in which stress responsivity might be viewed is in terms of how long it takes a subject's physiology to stress and then to recover from this increased arousal. One might argue for example that becoming physiologically aroused during stress is healthy and adaptive whereas staying aroused for a long period subsequent to the stressful event is unhealthy or nonadaptive.

On the basis of this physiological "recovery" rationale for differentiating stress responsiveness an additional analysis of the headache data was carried out. Subjects from the study were reclassified as being either EMG responsive, or, temperature responsive, according to time recovery scores. EMG recovery scores were calculated as the number of seconds following the presentation of the mental arithmetic task, that it took to return to the mean EMG level displayed during the three minute prestress relaxation phase.

Temperature recovery scores were calculated as the number of seconds which it took from the onset of the stress task, for each subject to recover 50 percent of the drop in temperature which had occurred during the stress period. The criteria used for calculating EMG and temperature recovery were adopted in order to conform to the general patterns of recovery obtained in the stress profiles. It was necessary to define recovery time in such a manner that subject variability in rate of recovery could be demonstrated within the five minute poststress phase when physiological measures were still being taken.

Reclassification of subjects into either the relatively high EMG responsivity group or the relatively high temperature responsivity group was carried out by the same median split procedure that was employed in the original classification of subjects.

The mean EMG recovery time for the reclassified EMG responsive group was 330 seconds. This score was significantly longer than the EMG recovery mean of 160 seconds obtained for the temperature responsive subjects ($t(46) = 3.53$, $p < .001$). The mean temperature recovery times were 313 seconds and 159 seconds for temperature responsives and EMG responsives respectively. The difference in these two time recovery means was also significant (Welch's $t(40) = -4.46$, $p < .001$).

Figure 6 illustrates minute-to-minute mean EMG values that were obtained for the two recovery groups during the

stress profiles. Each of the values are based on average ten-second EMG levels taken at the minutes indicated on the graph. It can be seen that the EMG responsive group that took longer to recover from stress, also demonstrated considerably more change in EMG from relaxation to stressing. However, it can also be noted that the EMG responsive group had lower EMG levels during the eyes closed relaxation phase than did the temperature responsive group. The mean EMG level for the EMG responsive group over the three minute eyes closed relaxation phase was 1.59 microvolts; the mean EMG level for the temperature responsive group during this phase was 2.37 microvolts. An independent t test was conducted on these means and the difference was found to be significant (Welch's $t(37) = -2.12$, $p < 0.05$). Apparently, the differentiation of subjects according to EMG and temperature recovery scores had the additional, inadvertent effect of identifying two groups of subjects who differed in basal frontal EMG. It is important to consider that the group of subjects classified as EMG responsive may have demonstrated slower recovery to their prestress EMG levels because these basal levels were so low. The remaining subjects, on the other hand, did not stress very far beyond their high basal EMG levels and consequently their frontal EMG responses recovered to the basal means rather quickly. It is possible that the high EMG group mean during relaxation, and the flat curve through the course of stressing, indicates that these subjects tended to

be in a chronic state of stress or arousal throughout the stress profile session.

Figure 7 illustrates minute-to-minute mean temperature values that were obtained for the two recovery groups during the stress profiles. The graph suggests that the temperature responsive subjects who were slow to recover from stress, also responded with a greater drop in temperature than did the EMG responsive group. The difference between the mean basal temperatures of the two groups in the three minute relaxation period prior to stressing was not significant.

**Summary**

The use of the median split procedure to reclassify subjects into two groups with differential EMG and temperature psychophysiological stress-recovery patterns was successful. Mean comparisons of EMG changes and temperature changes for the two responsivity groups indicated that one group demonstrated relatively long mean EMG responsivity time with relatively short temperature responsivity, while the other group presented a profile of relatively long temperature responsivity time with relatively brief EMG responsivity. In addition it was found that the EMG responsive group had a significantly lower basal mean EMG prior to stressing than the basal EMG mean displayed by the temperature responsive group.

**Daily Headache Record**

Table 2 (see next page) indicates the mean headache intensity levels in pretreatment, treatment and

posttreatment for the two recovery groups and the three treatment groups combinations. Figure 8 presents this data graphically. The interaction between treatments and groups with respect to headache reductions was tested by a two factor Analysis of Variance. Decrease in headache intensity from pretreatment to posttreatment was used as the numeric. The results indicated that the interaction was statistically significant $F(2,49) = 3.92$, $p < .05$. This interaction is presented graphically in Figure 9. Separate Scheffe post hoc comparisons were conducted on the two groups within each of the three treatments included in the study. Significant differences in headache reduction means were obtained between EMG and temperature responsivity subjects in each of the three treatments: *Sch* $F(5,13) = 1.449$, $p < .01$ for responsivity groups in EMG treatment; *Sch* $F(5,10) = 1.712$, $p < .01$ for responsivity groups in temperature treatment; and *Sch* $F(5,14) = 1.385$, $p < .01$ for responsivity groups in combined treatment.

The post hoc analyses indicated that EMG biofeedback produced more headache reduction with subjects classified as temperature responsive than it did with subjects classified as EMG responsive. The same finding held true for the combined treatment subjects. In contrast however, temperature biofeedback proved to be more effective with EMG responsive subjects than with the subjects classified as temperature responsive. The finding that EMG treatment was most effective in reducing headaches among the temperature

Table 3

Mean Headache Levels Across Responsivity Groups, Treatments and Periods.

| Recovery Group | Treatment Mode | Period | | |
|---|---|---|---|---|
| | | Pretreatment | Treatment | Posttreatment |
| | | $T_1$ | $T_2$ | $T_3$ |
| EMG Responsive | EMG | 7.7 | 9.2 | 7.5 |
| | Temperature | 8.3 | 5.3 | 3.6 |
| | Combined | 10.2 | 9.8 | 9.0 |
| Temperature Responsive | EMG | 6.3 | 4.7 | 2.1 |
| | Temperature | 8.3 | 9.3 | 6.5 |
| | Combined | 14.4 | 7.2 | 5.4 |
| | Period Means (N=55) | 7.3 | 7.5 | 5.6 |

responsive subjects, whereas temperature biofeedback was more effective among the EMG responsive subjects is further reinforced by the symmetrical pattern of headache reductions found with EMG and temperature treaments in the EMG responsive and the temperature responsive groups. This pattern is evident in Figure 8.

At first blush the interaction pattern between treatments and responsivity groups appears to contradict the original hypothesis that matching biofeedback treatment modalities with psychophysiological reactivity groups would optimize treatment effects. However, the pattern of results obtained must be considered in terms of the EMG and temperature stress profile data that were analyzed earlier. The profile analysis indicated that the group labelled as EMG responsive actually had a significantly lower basal EMG mean than the group labelled as temperature responsive. The stress profile data and the headache treatment results taken together indicated that chronically high basal EMG subjects with flat EMG and responsive temperature profiles during stress testing were the subjects who benefited most from EMG biofeedback treatment for their headaches. On the other hand subjects with low basal EMG who demonstrated a resposive EMG and a flat temperature profile during stress testing were more suited for temperature training.

In order to check the possibility that profile responsivity itself was unimportant in the prediction of biofeedback treatment results and that basal EMG and

temperature levels were the main factor involved, a separate median split classification was conducted. Subjects' EMG and temperature levels during the three minute relaxation period prior to stressing were used to produce two groups. One of these groups was characterized by high basal EMG and high basal temperature; the other group was characterized by low basal EMG and low basal temperature. A two-factor Analysis of Variance on the pretreatment to posttreatment headache reductions among the two groups and three treatments resulted in no significant interactions.

Although the interaction between EMG and temperature treatments with responsivity groupings has been elaborated upon, it remains to consider the differences in heachache change found between responsivity groupings who received combined biofeedback treatment. Figure 8 illustrates that a confounding aspect in the interpretation of the combined biofeedback effects is the fact that the pretreatment headache level of the temperature responsive subjects was considerably higher than the level obtained for the EMG responsive subjects. However, this confound may not account for the whole difference between responsivity groups as the temperature responsive subjects actually ended up with less headache in posttreatment than did the EMG responsive subjects.

**Summary**

The pattern of headache reductions from pretreatment to posttreatment among the two responsivity groups andthe

three treatments resulted in a statistically significant interaction effect. Frontal EMG biofeedback was more effective with subjects classified into the temperature responsive group than with subjects classified into the EMG responsive group; digit temperature biofeedback was more effective with subjects classified as EMG responsive than with subjects who were classified as temperature responsive; combined EMG and temperature biofeedback was more effective in reducing headaches with subjects classified as temperature responsive than with subjects classified as being EMG responsive.

## C. Biofeedback Training Data

### Reactivity Groupings

Frontal EMG levels and finger temperature levels were computed over the course of training to see if differential learning curves could be discerned among the treatments by groups combinations of subjects. EMG and temperature levels were sampled during the last five minutes of each training session because it was this interval in which subjects practiced relaxation in the absence of feedback during sessions five through nine. The EMG and temperature values computed during the five minutes were a mean of the five minute-by-minute ten second averages produced by the data acquisition unit. In addition, means were computed for the five minute eyes-open and the three minute eyes-closed relaxation phases from the stress profile sessions that were

held prior to treatment. These means computed from the stress profile sessions were also based on the ten second averages collected each minute.

A two (groups) by three (treatments) by eleven (periods) repeated measures Analysis of Variance was performed on the EMG and temperature data. The EMG analysis indicated a statistically significant period effect in which Conservative $F(1, 42) = 16.83$, $p < .001$; No main effects were obtained for the groups factor or the treatments factor and no significant interaction effects were obtained. A summary table for the EMG Analysis of Variance appears in Appendix G. Seven subjects were excluded from this analysis in order to maintain an equal number of subjects in each treatments by groups cell combination. These were the same seven subjects who were excluded from the headache data analysis.

Figure 10 illustrates the five minute mean EMG levels obtained by each of the treatment groups at several points during the course of the treatment program. A sharp drop in EMG can be noted for all groups when subjects closed their eyes during the stress profile. Subjects were asked to keep their eyes open during actual treatment sessions. Post hoc contrasts were made to compare the eyes-closed stress profile mean and the session nine treatment mean for each of the three treatment groups. It was reasoned that the eyes closed stress profile mean would be a good unbiased pretreatment mean to contrast with treatment levelsbecause

it was the experimenter's impression that some subjects learned to partially close their eyes over the course of treatment. The ninth treatment session was selected as the contrast mean for the final treatment level because session nine was held two months after the last regular session and therefore session nine levels would to some extent be a measure of the durability of treatment effects. The post hoc contrasts indicated a significant drop in EMG level for the EMG treatment group where $F(1, 42) = 20.93$, $p < 0.001$; and a significant drop in EMG level for the combined treatment group where $F(1, 42) = 11.84$, $p < 0.001$. No significant drop in EMG level was obtained for the temperature treatment group.

The three-factor repeated measures Analysis of Variance that was conducted on the temperature data resulted in statistical significance for the main period effect and the three factor interaction, but no clinical meaningfulness could be given to these results. A complex pattern of temperature changes among the treatments, groups and periods indicated that some extraneous variables, possibly climate or medication effects, must have contaminated the results. Figure 11 illustrates the mean temperature levels during the last five minutes of several of the treatment sessions. These means are for each of the three treatment groups with values for the reactivity groups collapsed. One may conclude from this graph that hand temperature for all groups was higher during training than it had been during the stress

profile session. No meaningful differentiation can be made among the groups.

**Recovery Groupings**

The biofeedback training data and the stress profile data analyzed above were reanalyzed with subjects grouped according to the reclassification that had been conducted on the bases of stress recovery time. The mean frontal EMG levels during the eyes closed relaxation phase of the stress profiles and the last five minutes of several training sessions are presented in Figure 12. The graph illustrates the elevated EMG levels demonstrated by the temperature responsive subjects during the stress profile session. A two (groups) by three (treatments) Analysis of Variance was conducted on the differences in means from the basal EMG levels obtained during the stress profile to the levels obtained during session nine. No significant main effects or interaction effects were obtained. However, a trend for significance was indicated on the responsivity groups factor: $F(1, 49) = 3.24$, $p < .08$. A summary table for this analysis appears in Appendix G.

Figure 13 illustrates the mean temperature levels obtained during the eyes closed relaxation phase of the stress profiles and the last five minutes of several training sessions, for each of the three treatments by two groups combination of subjects. No Analysis of Variance was conducted because of the data contamination mentioned above. The graphical trends in means indicate higher temperatures

during training sessions than were evidenced in the stress profile relaxation segment. No differences in temperature pattern can be discerned among the treatments and groups.

Figure 1

Reactivity t scores. Each dot represents a subject's position relative to all other subjects in terms of EMG and temperature stress-reactivity scores. The diagonal line indicates the division of subjects into two reactivity groups.

FIGURE 1

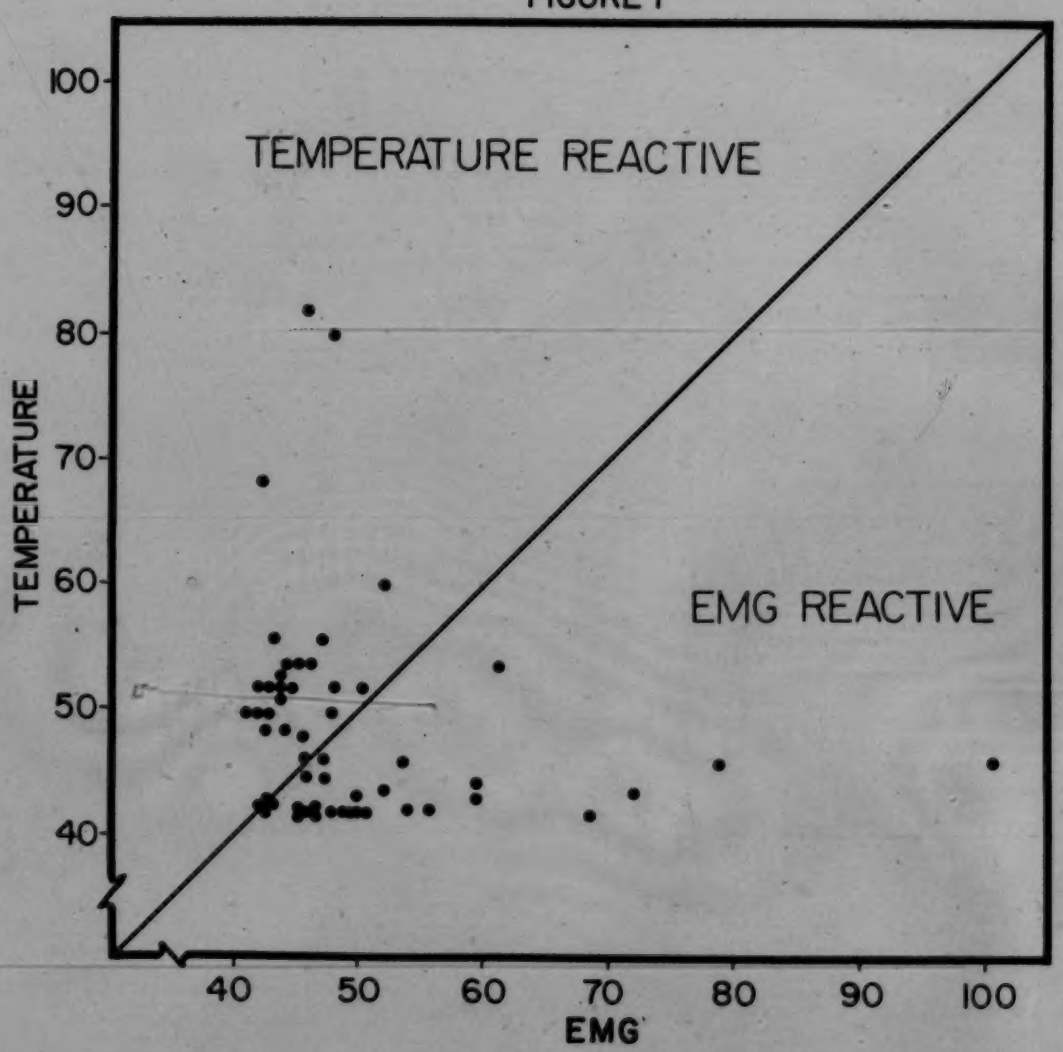

TEMPERATURE REACTIVE
EMG REACTIVE
TEMPERATURE
EMG
100
90
80
70
60
50
40
40
50
60
70
80
90
100

Figure 2

Mean EMG levels of the two stress reactivity groups during stress profile phases of relaxation, stress, and recovery; with eyes open (E.O.) or eyes closed (E.C.). EMG values plotted were 10 second averages at each of the minutes indicated.

Figure 2

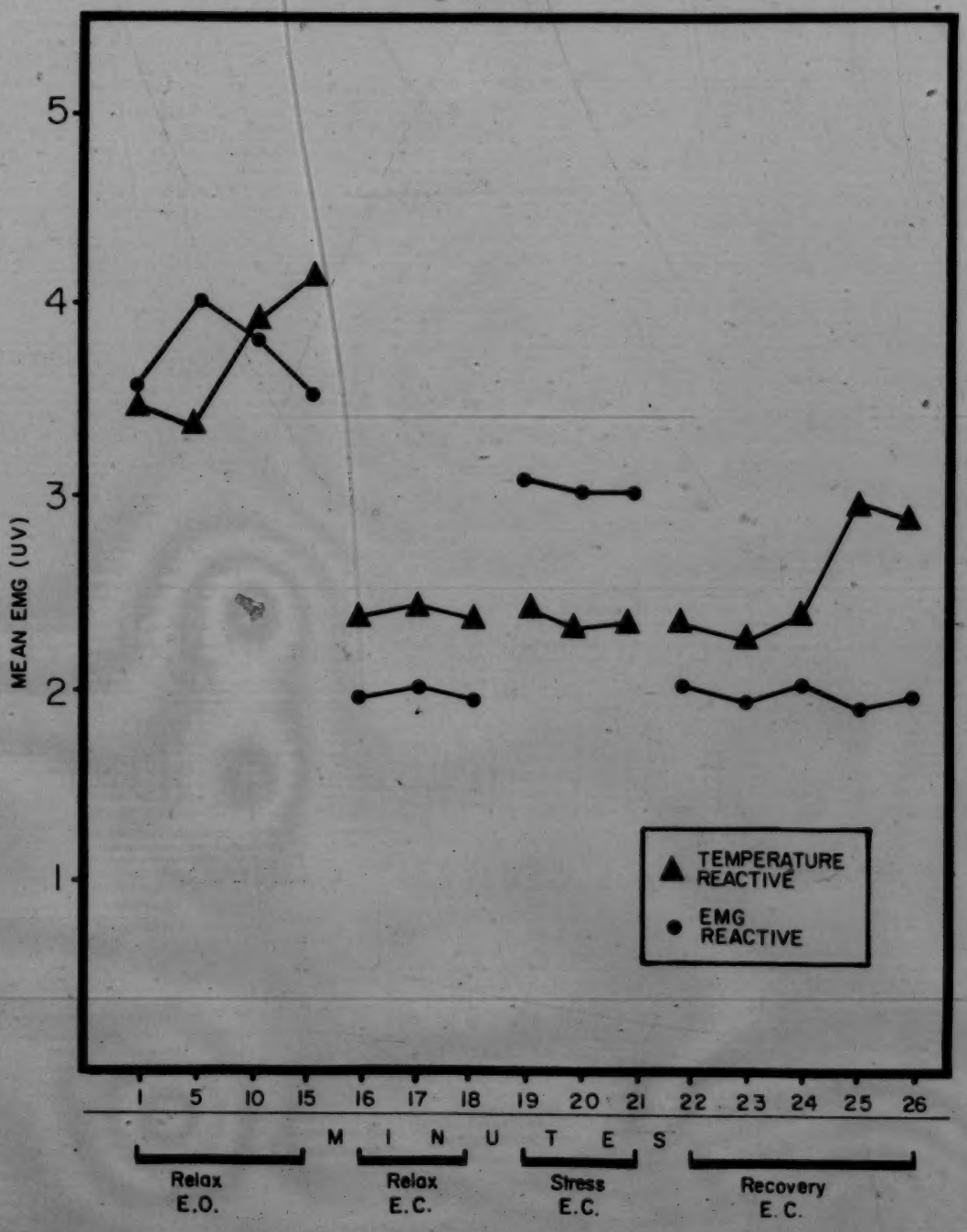

MEAN EMG (UV)
5
4
3
2
1
TEMPERATURE REACTIVE
EMG REACTIVE
1 5 10 15 16 17 18 19 20 21 22 23 24 25 26
MINUTES
Relax E.O.
Relax E.C.
Stress E.C.
Recovery E.C.

Figure 3

Mean temperature levels of the two stress reactivity groups during stress profile phases of relaxation, stress and recovery; with eyes open (E.O.) or eyes closed (E.C.). Temperature values plotted were 10 second averages at each of the minutes indicated.

Figure 3

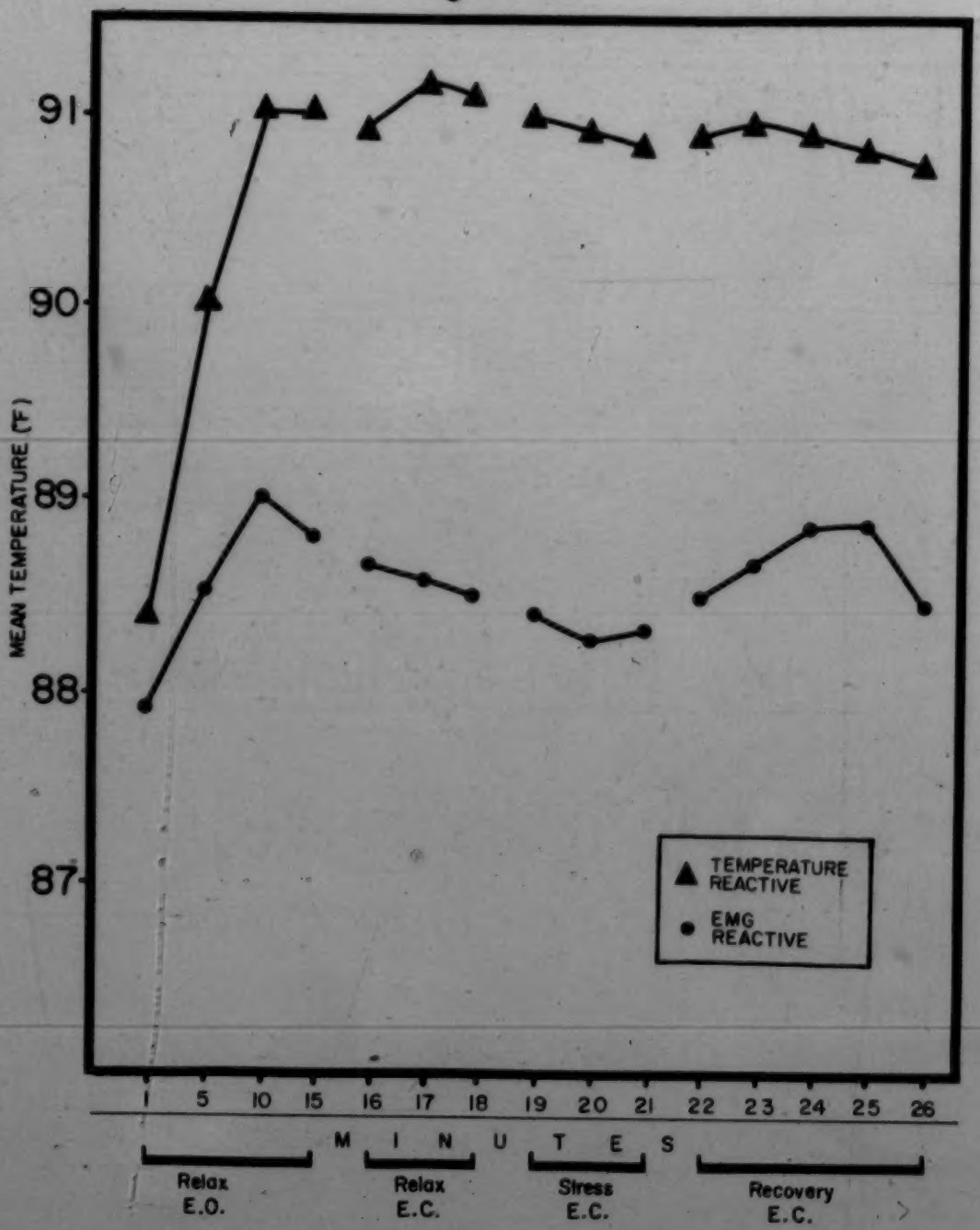

91
90
89
88
87
MEAN TEMPERATURE (°F)
TEMPERATURE REACTIVE
EMG REACTIVE
1 5 10 15 16 17 18 19 20 21 22 23 24 25 26
MINUTES
Relax E.O.
Relax E.C.
Stress E.C.
Recovery E.C.

Figure 4

Diagram for experimental design. Cell entries were group mean scores. The dependent variable was mean headache rating per day.

FIGURE 4

| TREATMENT GROUPS | | EXPERIMENTAL PERIOD Factor C | | |
|---|---|---|---|---|
| Factor A | Factor B | PRE TREATMENT $T_1$ | TREATMENT $T_2$ | POST TREATMENT $T_3$ |
| EMG Reactive | EMG Treatment | n=10 | n=10 | n=10 |
| | Temperature Treatment | n=10 | n=10 | n=10 |
| | Combined Treatment | n=10 | n=10 | n=10 |
| Temperature Reactive | EMG Treatment | n=10 | n=10 | n=10 |
| | Temperature Treatment | n=10 | n=10 | n=10 |
| | Combined Treatment | n=10 | n=10 | n=10 |

Figure 5

Mean headache intensity levels for the three treatments (EMG, temperature and combined), two groups (EMG reactive and temperature reactive) and three periods ($T_1$ = pretreatment, $T_2$ = treatment, $T_3$ = posttreatment. N=48.

FIGURE 5

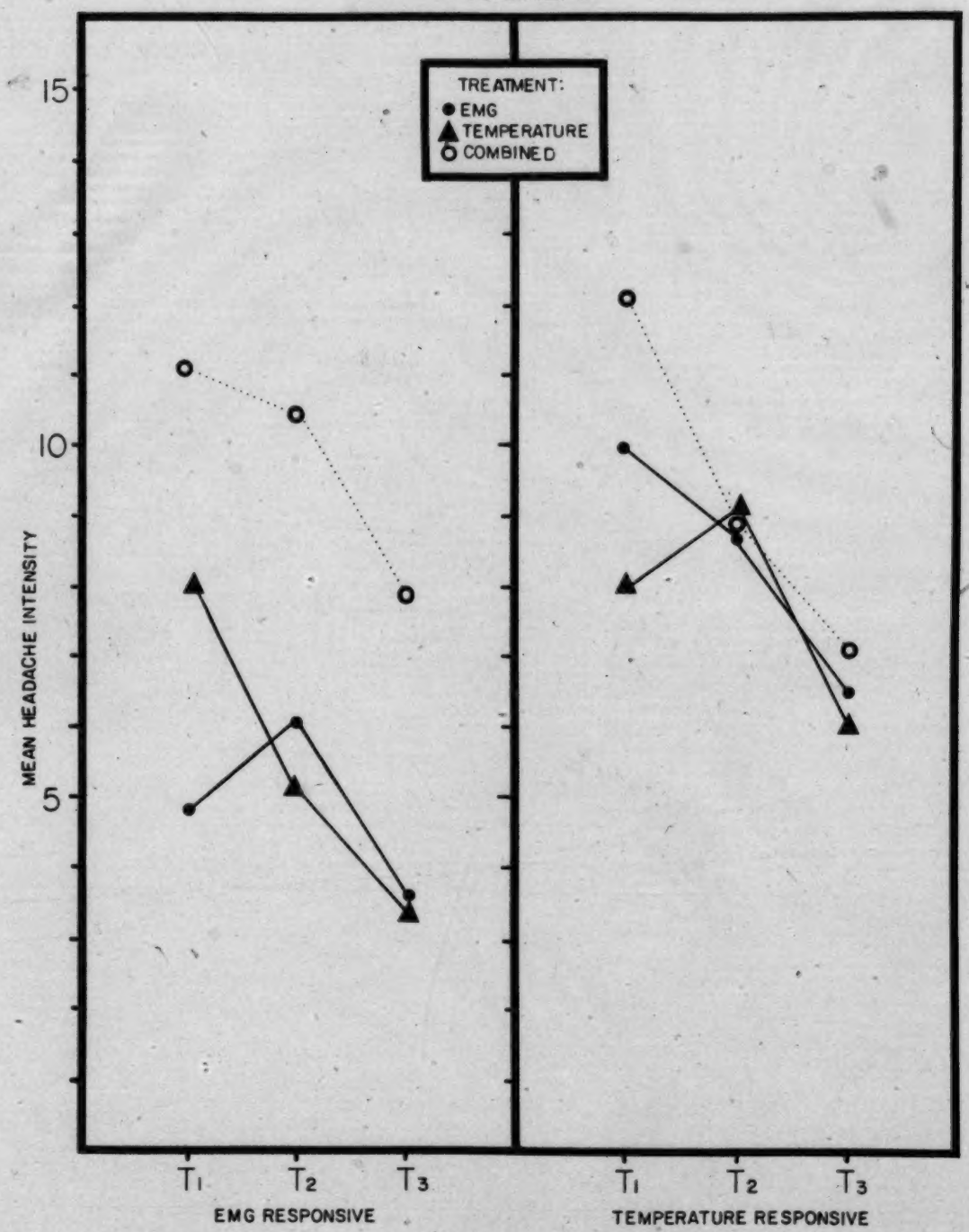

TREATMENT:
EMG
TEMPERATURE
COMBINED
15
10
5
MEAN HEADACHE INTENSITY
T1
T2
T3
EMG RESPONSIVE
T1
T2
T3
TEMPERATURE RESPONSIVE

Figure 6

Mean EMG levels of the two reclassified stress responsivity groups during stress profile phases of relaxation, stress, and recovery; with eyes open (E.O.) or eyes closed (E.C.). EMG values plotted were 10 second averages at each of the minutes indicated.

Figure 6

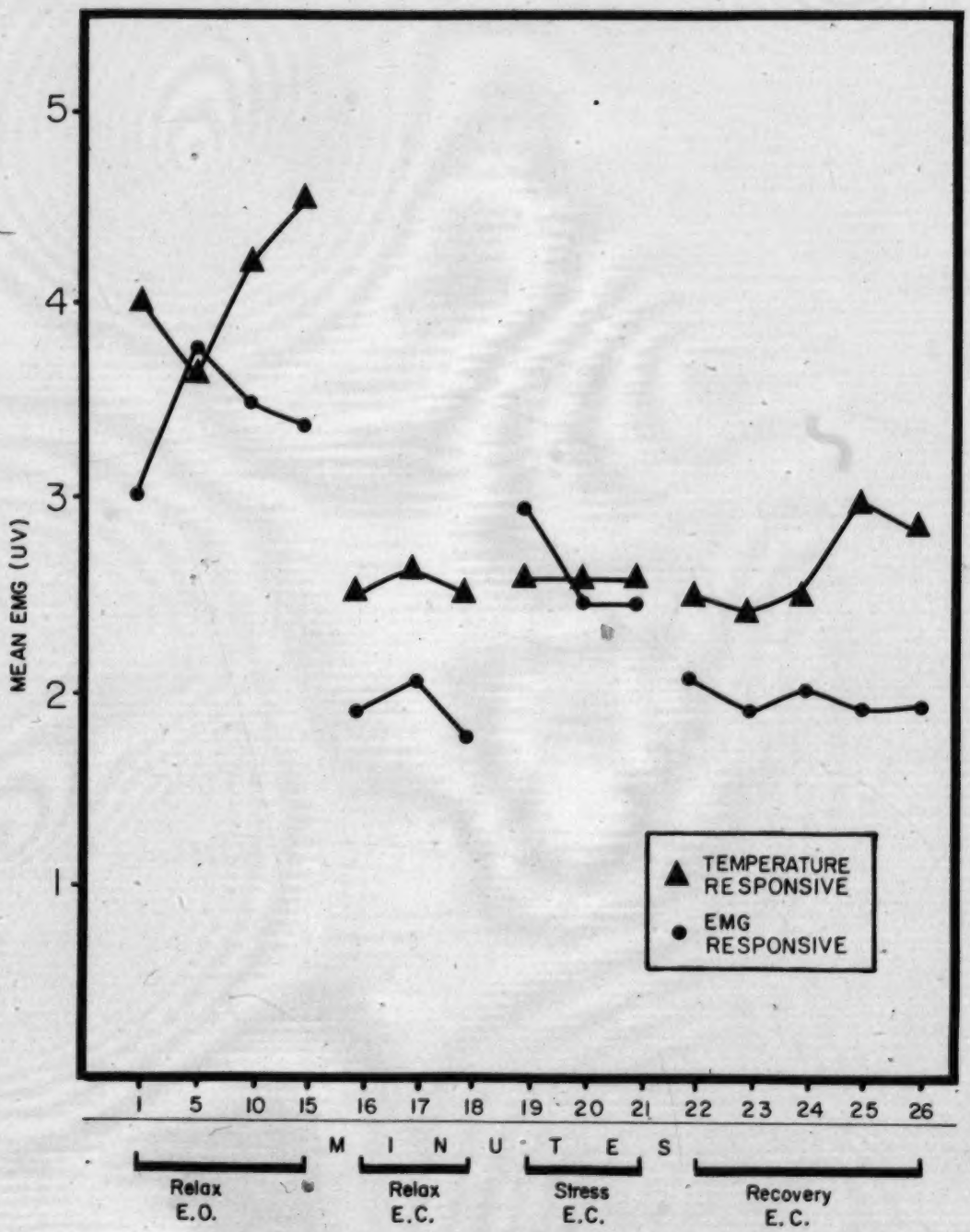

MEAN EMG (UV)
5
4
3
2
1
TEMPERATURE RESPONSIVE
EMG RESPONSIVE
1 5 10 15 16 17 18 19 20 21 22 23 24 25 26
MINUTES
Relax E.O.
Relax E.C.
Stress E.C.
Recovery E.C.

Figure 7

Mean temperature levels of the two reclassified stress responsivity groups during stress profile phases of relaxation, stress, and recovery; with eyes open (E.O.) or eyes closed (E.C.). Temperature values plotted were 10 second averages at each of the minutes indicated.

Figure 7

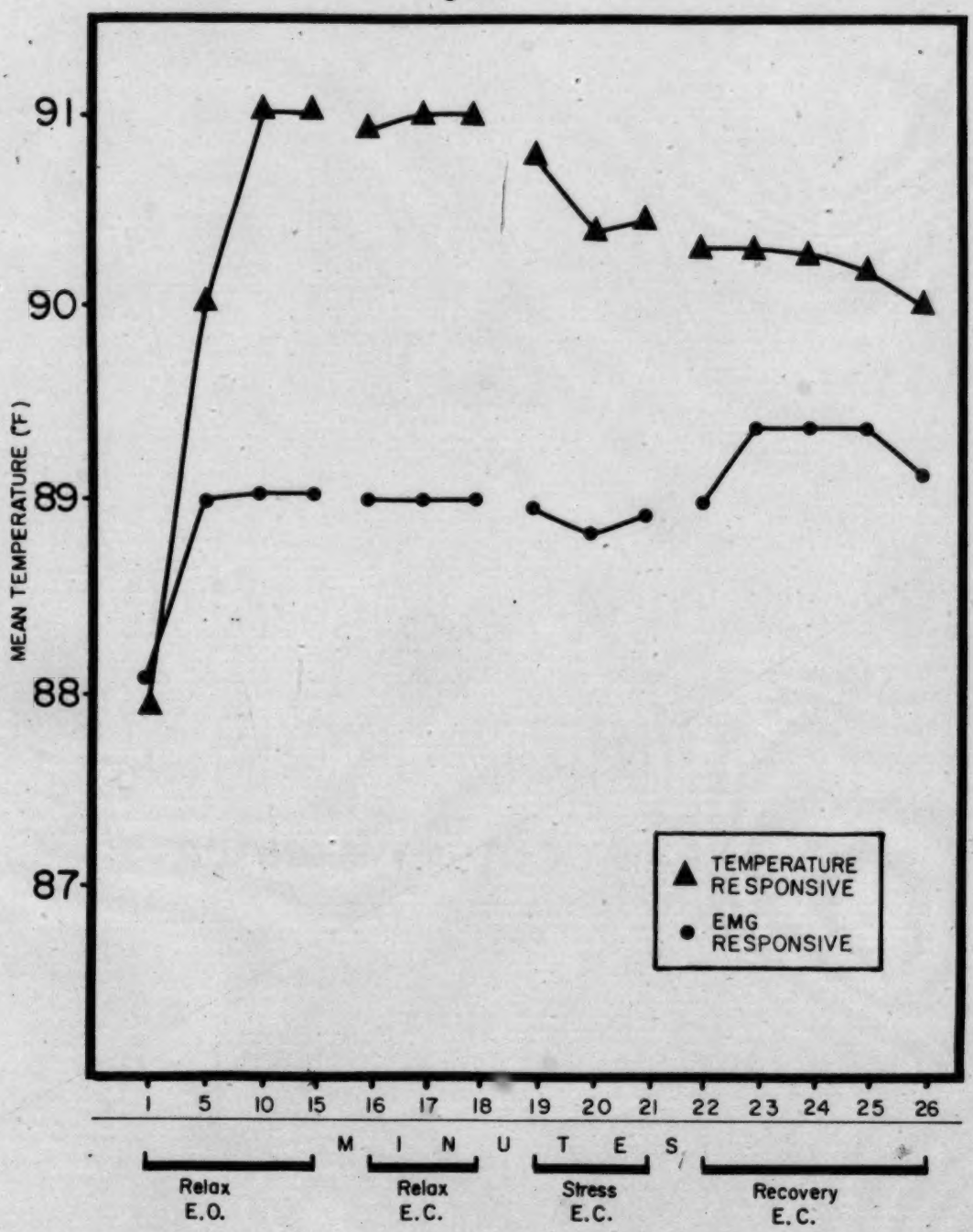

91
90
89
88
87
MEAN TEMPERATURE (°F)
TEMPERATURE RESPONSIVE
EMG RESPONSIVE
1 5 10 15 16 17 18 19 20 21 22 23 24 25 26
MINUTES
Relax E.O.
Relax E.C.
Stress E.C.
Recovery E.C.

Figure 8

Mean headache intensity levels for the three treatments (EMG, temperature, and combined), two reclassified groups (EMG responsive and temperature responsive) and three periods ($T_1$ = pretreatment, $T_2$ = treatment, $T_3$ = posttreatment). N=55.

FIGURE 8

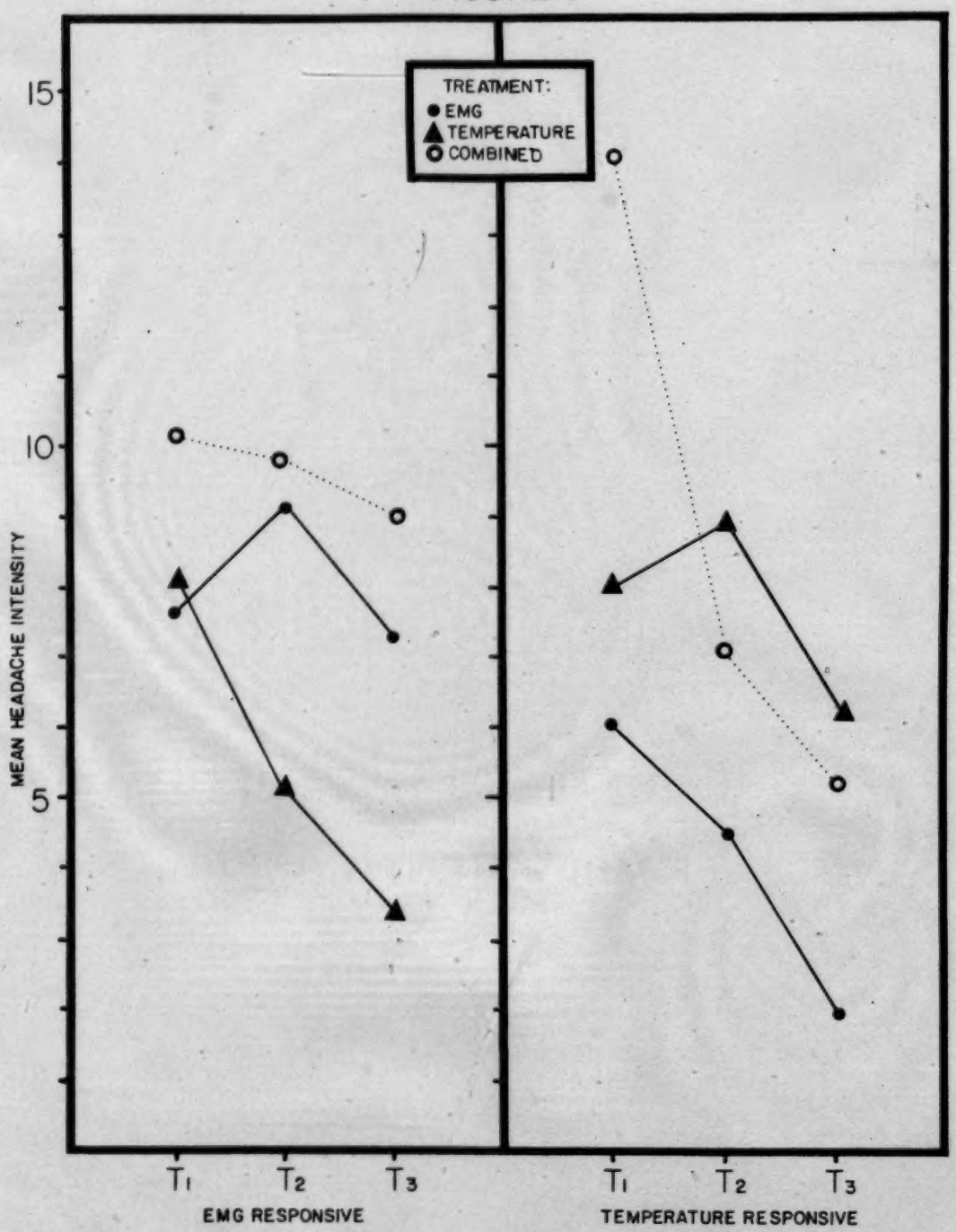

TREATMENT:
EMG
TEMPERATURE
COMBINED
15
10
5
MEAN HEADACHE INTENSITY
T1
T2
T3
EMG RESPONSIVE
T1
T2
T3
TEMPERATURE RESPONSIVE

Figure 9

Mean headache reduction scores from pretreatment to posttreatment for the three treatments (EMG, temperature and combined) and two classification groups (EMG responsive and temperature responsive).

# FIGURE 9

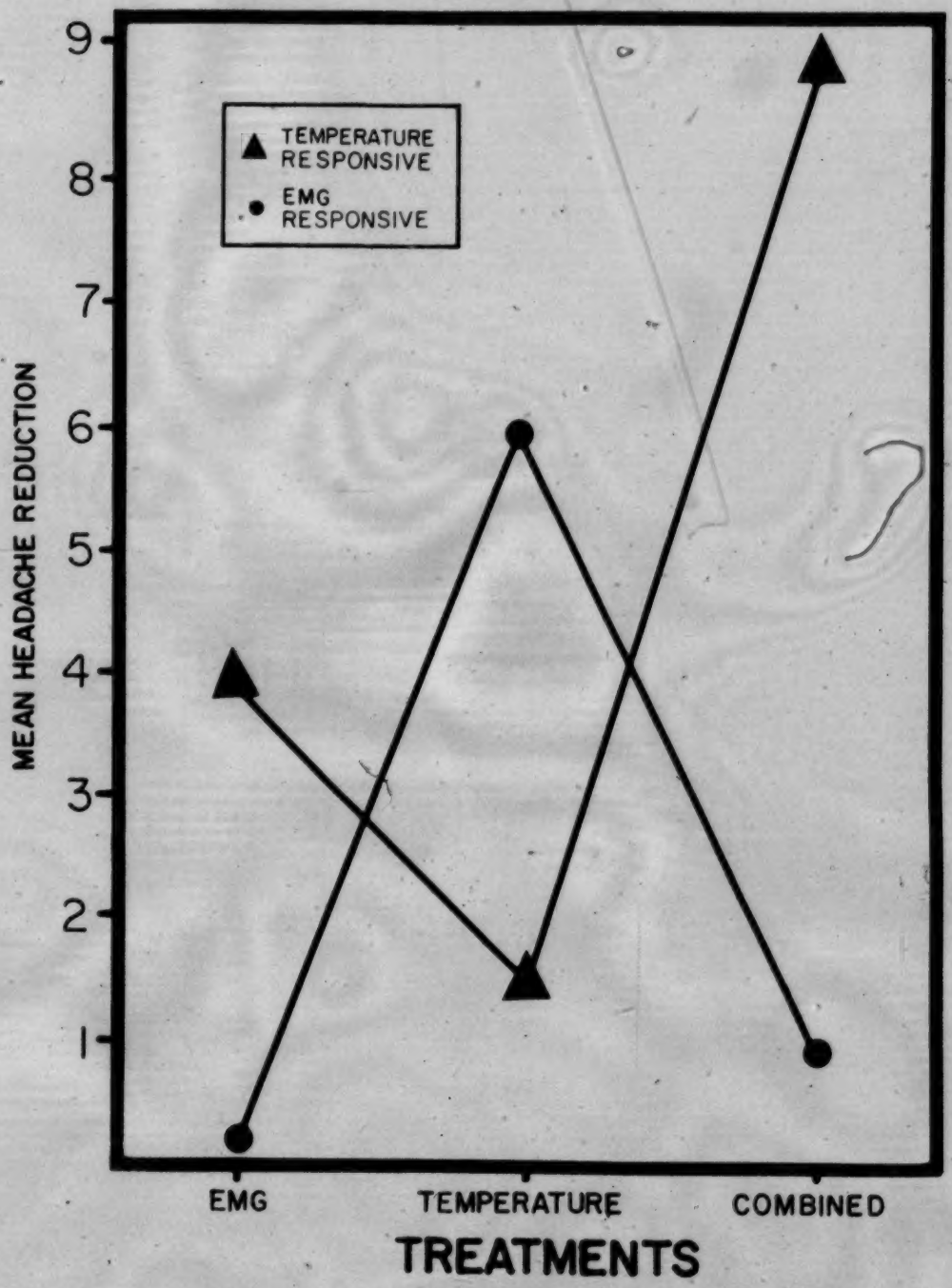

TEMPERATURE RESPONSIVE
EMG RESPONSIVE
MEAN HEADACHE REDUCTION
9
8
7
6
5
4
3
2
1
EMG
TEMPERATURE
COMBINED
TREATMENTS

Figure 10

Mean EMG levels for the three treatment groups during the eyes open (E.O.) and the eyes closed (E.C.) relaxation phases of the stress profile session, and the final five minutes of treatment sessions 3, 5, 7, and 9. Relaxation practice in the absence of feedback was in effect during the graphed intervals for sessions 5, 7, and 9.

FIGURE 10

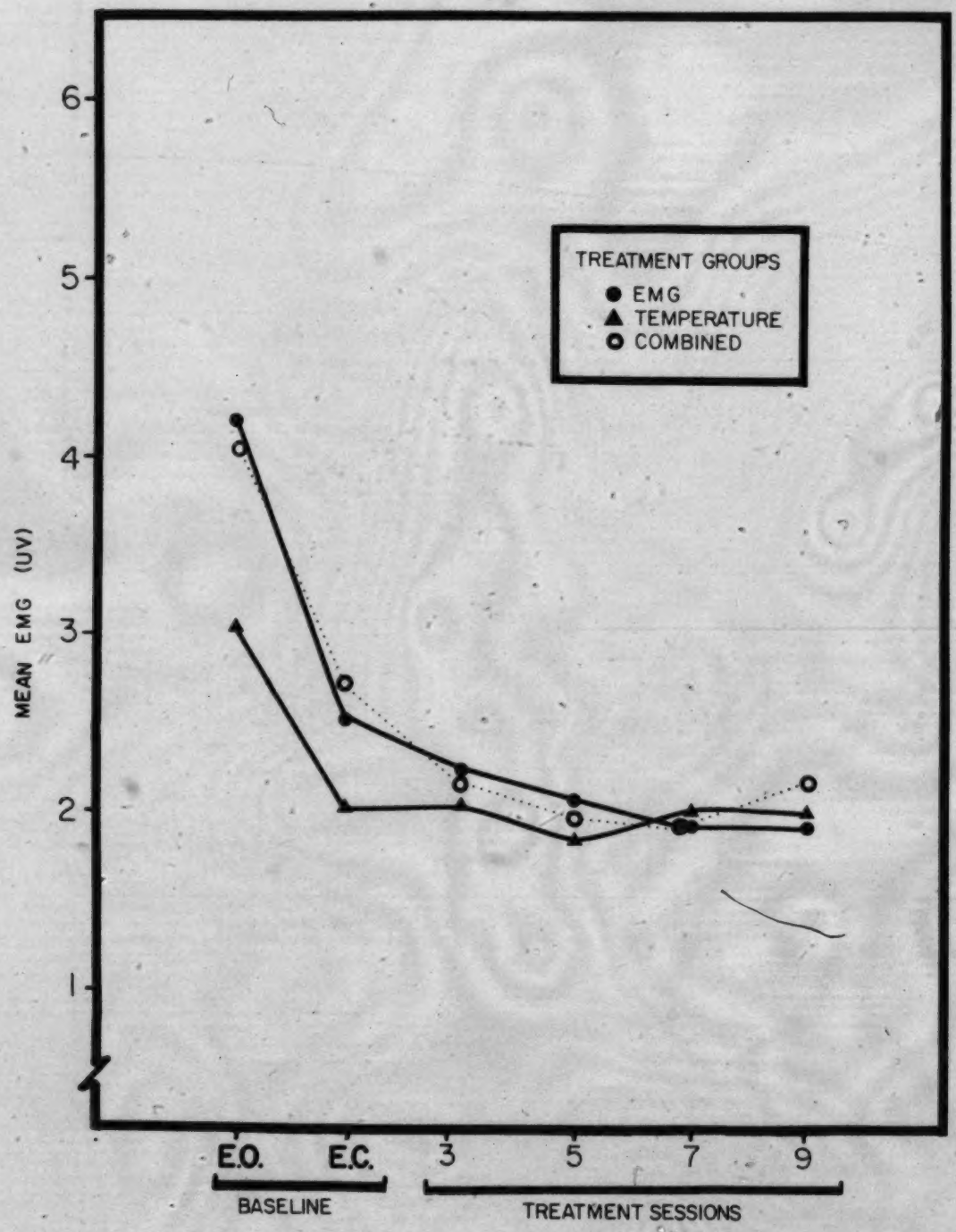

TREATMENT GROUPS
EMG
TEMPERATURE
COMBINED
MEAN EMG (UV)
6
5
4
3
2
1
E.O.
E.C.
3
5
7
9
BASELINE
TREATMENT SESSIONS

Figure 11

Mean temperature levels for the three treatment groups during the eyes closed relaxation phase of the stress profile session, and the final five minutes of treatment sessions 3, 5, 7, and 9. Relaxation practice in the absence of feedback was in effect during the graphed intervals for sessions 5, 7, and 9.

FIGURE 11

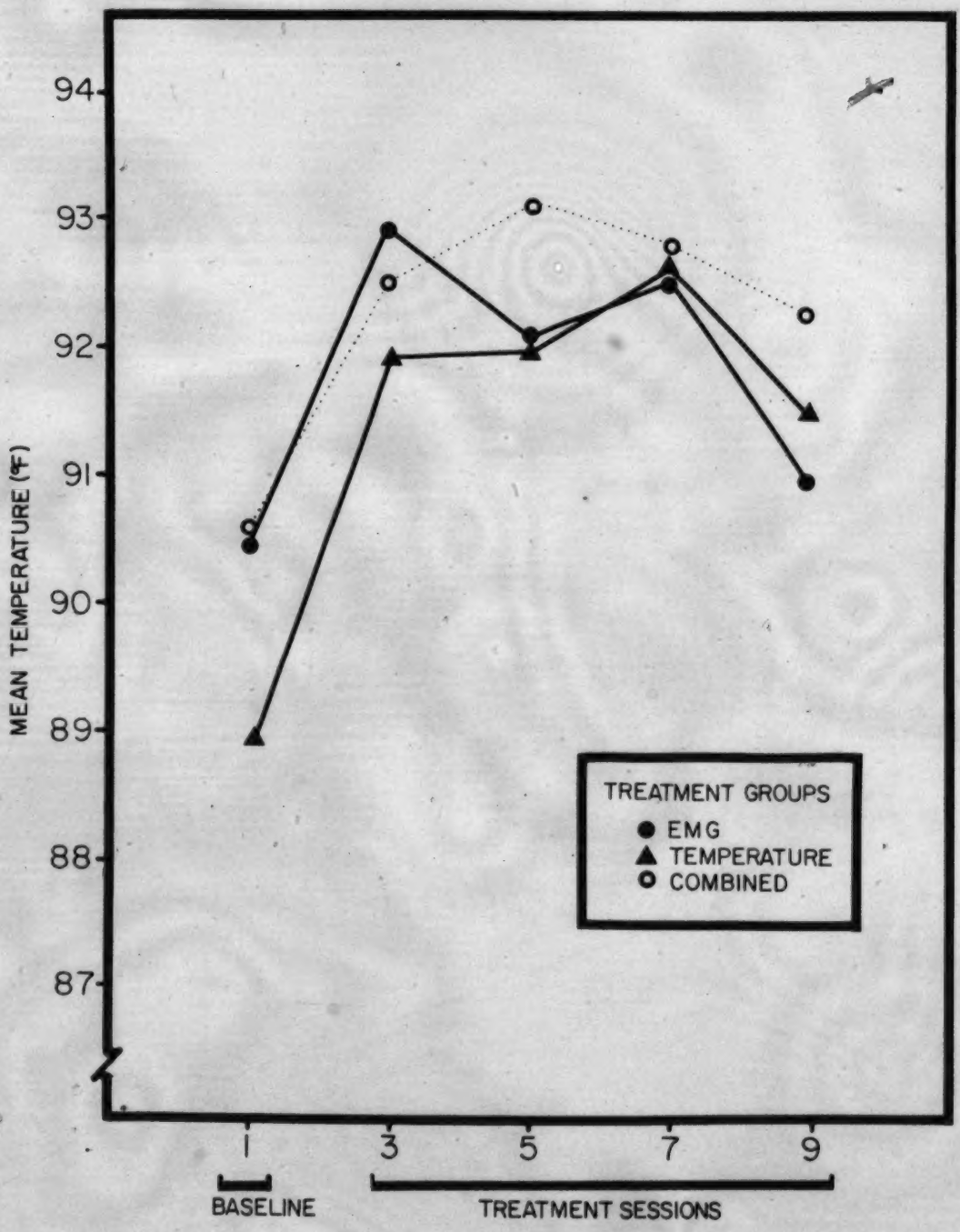

94
93
92
91
90
89
88
87
MEAN TEMPERATURE (°F)
1
3
5
7
9
BASELINE
TREATMENT SESSIONS
TREATMENT GROUPS
EMG
TEMPERATURE
COMBINED

Figure 12

Mean EMG levels for the three treatments and the two responsivity groups during the eyes closed relaxation phase of the stress profile session (B = Baseline), and the final five minutes of treatment sessions, 3, 5, 7, and 9. Relaxation practice in the absence of feedback was in effect during the graphed intervals for sessions 5, 7, and 9.

FIGURE 12

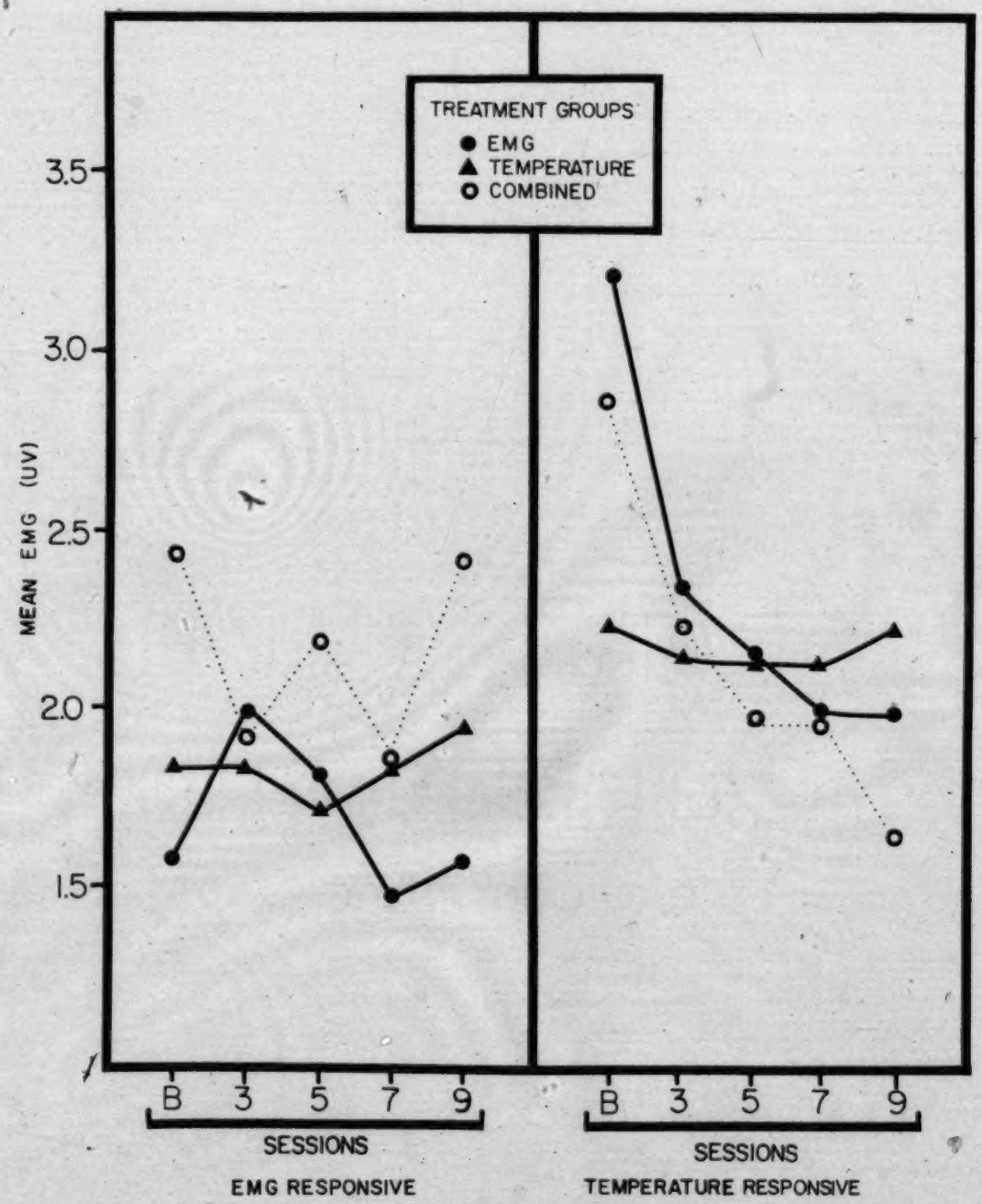
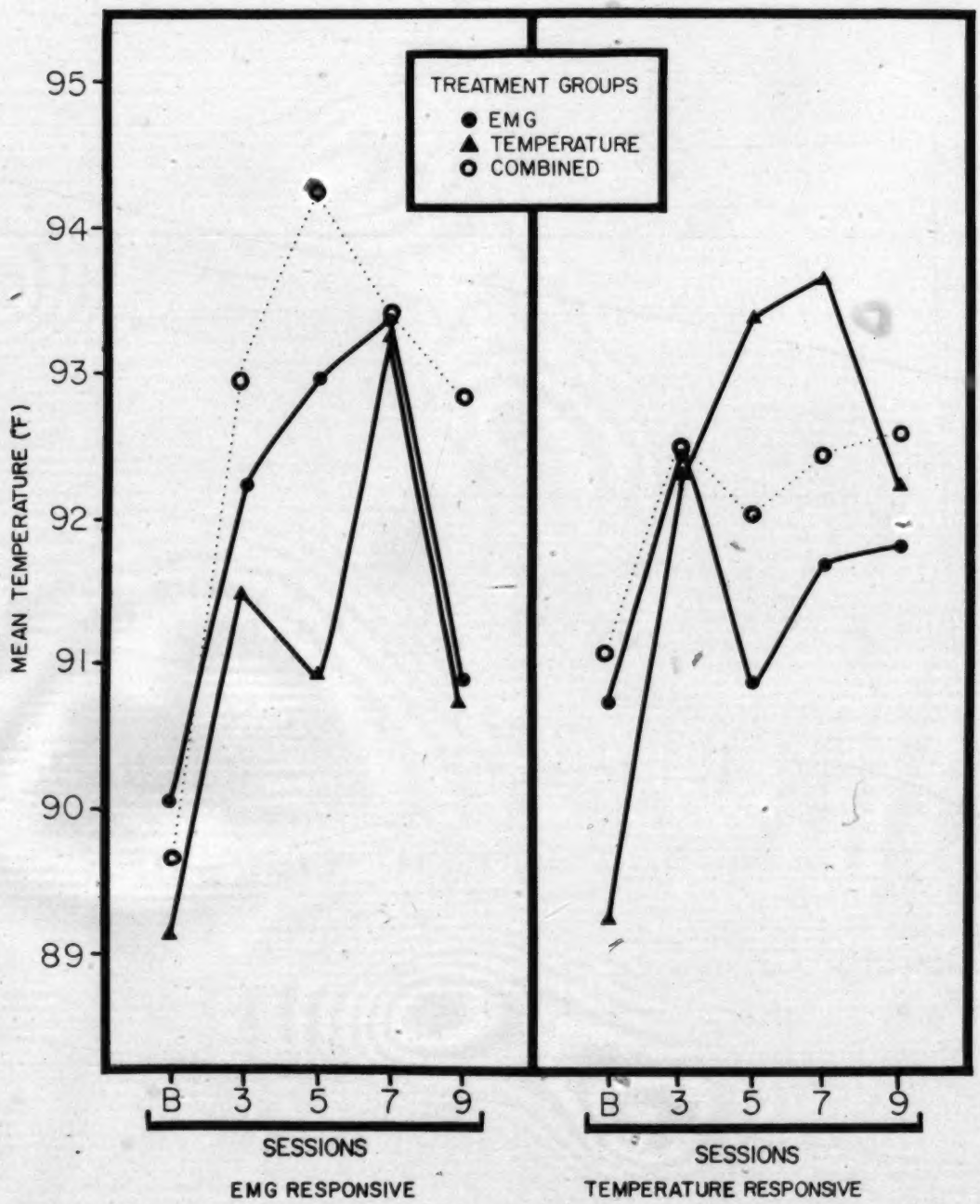

TREATMENT GROUPS
EMG
TEMPERATURE
COMBINED
MEAN EMG (UV)
3.5
3.0
2.5
2.0
1.5
B 3 5 7 9
SESSIONS
EMG RESPONSIVE
B 3 5 7 9
SESSIONS
TEMPERATURE RESPONSIVE

Figure 13

Mean temperature levels for the three treatments and two responsivity groups during the eyes closed relaxation phase of the stress profile session (B = baseline), and the final five minutes of treatment sessions 3, 5, 7, and 9. Relaxation practice in the absence of feedback was in effect during the graphed intervals for sessions 5, 7, and 9.

FIGURE 13


TREATMENT GROUPS
EMG
TEMPERATURE
COMBINED
MEAN TEMPERATURE (°F)
95
94
93
92
91
90
89
B 3 5 7 9
SESSIONS
EMG RESPONSIVE
B 3 5 7 9
SESSIONS
TEMPERATURE RESPONSIVE

# V. Discussion

## Major Findings

The results of this study have provided empirical evidence that EMG biofeedback, hand-temperature biofeedack, and combined EMG and hand temperature biofeedback were differentially effective in reducing headaches among a group of subjects with migraine symptoms. The differential effectiveness among the three types of biofeedback training was only apparent when migraine subjects were divided into two groups according to psychophysiological patterns of stress recovery. It was found that frontal EMG biofeedback and combined EMG and hand-temperature biofeedback were more effective with subjects who demonstrated relatively slow temperature recovery from stress, and relatively fast EMG recovery from stress; temperature biofeedback was more effective in reducing headaches with subjects who demonstrated relatively slow EMG recovery and relatively fast temperature recovery. No differences among the three types of biofeedback could be discerned when subjects were grouped according to how much their EMG and temperature levels changed following the stress task.

Analyses of the stress profile data by which subjects were divided into groups indicated that the group of subjects who had relatively fast EMG recovery times also had a significantly higher mean level of basal EMG than the group of subjects who had relatively slow EMG recovery. The characteristic features then of the group who benefitted

most from EMG and combined biofeedback were a flat EMG profile during stressing, a high basal EMG level prior to stressing and a responsive temperature profile which was slow to recover subsequent to stressing. The characteristic features of the group of subjects who benefitted most from temperature biofeedback were a flat temperature profile during stressing, a low basal EMG level prior to stressing and a responsive EMG profile which was relatively slow to recover following the mental stressor.

The finding of differential effectiveness in the application of the three treatment regimes to the two responsivity groupings has strong theoretical implications. Considerable research has been conducted to verify that several forms of biofeedback and relaxation training are superior to placebo control procedures in reducing migraine. To date however little research has been conducted to clarify whether some types of biofeedback or relaxation training are more effective with some individuals than with others (Blanchard, Andrasik, Ahles, Teders, & O'Keefe, 1980). Clearly more research is needed in this direction in order that treatment procedures which will maximize clinical results may eventually be selected for individual clients. The results obtained in the present study indicate that stress profile testing is a promising avenue for such needed investigation. In particular, basal levels of physiological activity, duration of physiological activation subsequent to stressing and measures of profile flatness from relaxation

to stressing appear to be important variables in the prediction of biofeedback treatment effects with migraine patients.

The size of the sample used in this study, and the median split procedure for dividing subjects into groups, are two important factors which limit the immediate practical implications of the results obtained. Many more subjects would be required before a researcher could attain adequate norms for stress profile data. At this point there is no way of telling what proportion of a population of subjects would maximally benefit from any one form of biofeedback treatment. Furthermore, it must be kept in mind that the treatment for migraines which was provided in this study was limited to nine half-hour sessions. It is possible that significant treatments-by-groups interaction effects would not have been obtained if the author had conducted more or even fewer treatment sessions. A final qualification of the results which is important to mention is the fact that the post-treatment follow-up period was one-month in duration. One might argue that more clinical significance would be obtained if this period was lengthened.

**The Research Questions**

The design of this investigation was developed to consider four specific questions. Each of these will now be addressed.

*Questions One.* Is digit temperature biofeedback in combination with EMG biofeedback more effective in the

management of migraine than either digit temperature biofeedback alone, or EMG biofeedback alone?

The three factor repeated measures Analyses of Variance which was conducted on the headache data indicated that no statistically significant interactions occurred between treatments and periods. In other words no differences were found in the effectiveness of the three treatments when stress profile groupings were collapsed. One must be careful not to interpret these findings to mean that no differences actually existed in the effectiveness of each of the three treatment groups. A limitation of Analyses of Variance is the fact that this statistical approach is designed to test whether the null hypotheses of no differences should be rejected; the approach is not designed to measure the probability that obtained effects are not different. This point is particularly important when one considers the different baseline levels of headache which were obtained among the three treatment groups. Although the differences between groups were not found to be significantly different in the Analysis of Variance it can be seen in Figure 5 and Figure 8 that a sizeable difference existed between the baseline headache level for the combined group and the baseline levels for the other two treatment groups. This difference militates against very meaningful treatment comparisons between the combined treatment group and each of the other two groups.

Question One is best answered then by stating that the present study did not find the combined biofeedback treatment procedure to be superior to the EMG treatment or the temperature treatment when migraine subjects were considered as one undifferentiated group.

*Question Two*. Do EMG reactive subjects in EMG treatment and temperature reactive subjects in temperature treatment experience a greater reduction in migraines, than subjects with relatively low EMG reactivity who are given EMG treatment and subjects with relatively low temperature reactivity who are given temperature biofeedback treatment?

No significant differences were found among the treatments-by-groups combinations of subjects when the groupings of subjects was based on the differential amounts of change in EMG and temperature levels subsequent to stressing.

*Question Three*. Are differential biofeedback treatment effects more apparent when subjects are grouped according to criteria based on amount of physiological reactivity or when they are grouped according to duration of physiological reactivity?

Differential biofeedback treatment effects among the stress profile groupings of subjects was only apparent when the subjects were divided according to duration criteria of physiological reactivity. Furthermore, the pattern of differential biofeedback treatment effects which was obtained was not predicted prior to the study.

The author predicted that EMG treatment would be more effective with EMG responsive subjects and, temperature treatment would be more effective with temperature responsive subjects. In addition it was predicted that combined treatment would be equally effective with both responsivity groups. As outlined above the EMG and the combined biofeedback treatments were most effective with the temperature responsive subjects while the temperature biofeedback treatment was most effective with the EMG responsive subjects. Stress profile data provided some clues to the probable reasons for the results obtained among the two groups, and the EMG and temperature biofeedback treatment combinations. Interpretation of the differential effectiveness of the combined treatment between the EMG and temperature responsive groups is more difficult. It appears that combined EMG and temperature biofeedback was functionally similar to EMG biofeedback alone. Perhaps the subjects found the EMG feedback more salient or interesting than the temperature feedback and so they did not obtain much benefit from the temperature biofeedback component. In retrospect the EMG biofeedback which was included in combined treatment was provided auditorally and visually while temperature biofeedback was only provided visually. It is also worth noting that the EMG response tends to vary more than the temperature response on a second-to-second basis. Therefore, one might speculate that there is less tedium involved in attending to the EMG biofeedback signal

than there is when attending to the temperature signal. When they were given the choice the subjects may have preferred to attend to the EMG signal.

*Question Four*. What are some of the differential EMG and temperature performance characteristics among the various biofeedback and stress reactivity groupings of subjects?

No definitive differential EMG and temperature biofeedback performance characteristics were found among the treatments-by-groups combinations of subjects. Statistically significant interaction effects were not obtained in the Analyses of Variance that was conducted on the EMG data and temperature training data appeared to be contaminated by extraneous factors.

The main period effect which was found to be significant in the EMG data indicated an overall drop in EMG from the eyes open relaxation phase of the stress profile session to the treatment sessions. Much of this decrease can be attributed to adaptation effects which may have occurred as the subjects got used to the biofeedback instrumentation and the laboratory setting. Post hoc contrasts conducted on EMG means during eyes closed relaxation before treatment and eyes open relaxation in session nine indicated significant decreases for the EMG and combined treatment groups but no significant change for the temperature biofeedback group. These data suggest that some degree of physiological specificity in EMG assisted relaxation training may have

occurred. However, no definitive statement about such an effect can be made in the absence of a statistically significant interaction effect in the Analysis of Variance.

**Treatment Mechanisms**

The three biofeedback treatments employed in this study were differentially effective in reducing migraines among the stress recovery groups of subjects. The writer has interpreted these findings in terms of the logical suitability of each type of biofeedback for the differential patterns of stress profile which were found. Actual physiological data during biofeedback training did not provide clear evidence of differential learning rates among the treatments-by-groups combinations.

In the present study the evidence for differential biofeedback learning curves was sought during the final five minutes of treatment sessions. It was during this segment that clients practiced relaxation control in the absence of the feedback signal. In a recent study Bild and Adams (1980) have argued that the most accurate measure of learning in biofeedback is the ability to voluntarily control a response before beginning feedback in a session. Such measures require the subject to demonstrate what he can do without receiving biofeedback practice in advance during any one treatment session. In order to measure the subject's control prior to feedback an adaptation phase and a baseline phase would also be necessary. An obvious cost for such rigour is the increased time required to conduct a session.

Furthermore, it may be the case that subjects get some benefit out of biofeedback during the adaptation phase when physiological response levels typically are changing. This notion is probable in light of recent evidence which shows that the ability to discriminate changes in physiological response is a key element for clinical benefit (Jurish, Blanchard, Andrasik and Epstein; Note 6). Alternatively, Shein and Mandel (Note 7) have argued that standard deviation or minute-by-minute response variation is an important indicator of self-regulation in biofeedback. Obviously, more basic research in the area of biofeedback learning is needed in order to clear up these questions.

In the absence of data to substantiate that it was the physiological control skills learned in biofeedback that were responsible for migraine reduction, many alternative explanations are possible. The promotion of life style changes by biofeedback, expectancy for improvement, treatment credibility, and self-generated coping strategies are a few of the alternative factors which were covered earlier in the section on Therapeutic Intent contained in the Introduction. If such explanations are true then the results of the present study suggest that they were somehow related to the type of biofeedback treatment and the pattern of psychophysiological responsivity. For example, subjects with high basal EMG who were assigned to EMG treatment may have come to realize that they were chronically tense and thus took steps to change this situation. They may have

taken up jogging on a regular basis or they may have purposefully relaxed their muscles whenever they noticed their muscle tension in a stressful situation. Alternatively finding out that their headaches may be related to high levels of muscle tension and knowing that they were receiving biofeedback for muscle tension may have convinced some subjects that they did not have to anticipate the occurrence of so many headaches any longer. Consequently these subjects may have adapted a more carefree mental set and lifestyle. Endless possibilities exist. In fact many subjects made comments which suggested that such factors came into play to some degree. One person reported that subsequent to temperature biofeedback, when she noticed her hands were cold she would switch whatever household task she had been involved in to take on something more active. A couple of subjects reported that they had a tendency to clench their jaws and so they tried to catch themselves in the act in order to voluntarily stop. These changes in life style habits and activities may very well have had an effect on the incidence of migraines.

## REFERENCE NOTES


1. Packer, L.E. & Selekman, W.L. *Within subject controls in EMG and thermal biofeedback training: The baseline effect*. Unpublished manuscript, Adelphi University, 1977.

2. Turin, A. *Biofeedback for migraines*. Paper read at the Biofeedback Research Society, Monterey, California. February, 1975.

3. Hiebert, B.A. *A comparison of EMG feedback and alternative anxiety treatment programs*. Unpublished doctoral thesis. University of Alberta, 1979.

4. Libo, L.M. & Fehmi, L.G. *Cognitive strategies in biofeedback training of peripheral skin temperature*. Paper presented at the Biofeedback Society of America Eighth Annual Meeting, Orlando, 1977.

5. Hiebert, B.A. *Physiological response patterning: A review*. Paper presented to the Fourth Annual Meeting of the Biofeedback Association of Alberta. Edmonton, 1980.

6. Jurish, S., Blanchard, E.B., Andrasik, F. & Epstein, L.H. *Relationship between muscle discrimination ability and response to relaxation training in three kinds of headaches*. Paper presented at the Biofeedback Society of America Twelfth Annual Meeting, Louisville, 1981.

7. Shein, G.F. & Mandel, A.R. *Standard deviation as an indicator of self-regulation in biofeedback*. Paper presented at the Biofeedback Society of America Twelfth Annual Meeting, Louisville, 1981.

## REFERENCES

Ad Hoc Committee on Classification of Headache. Classification of headache. *Journal of the American Medical Association*, 1962, *179*, 717-718.

Adams, H.E., Feuerstein, M., & Fowler, J.L. Migraine headache: Review of parameters, etiology, and intervention. *Psychological Bulletin*, 1980, *87*, 217-237.

Adler, C.S. & Adler, S.M. Biofeedback - psychotherapy for the treatment of headaches: A 5-year follow-up. *Headache*, 1976, *16*, 189-191.

Andrasik, F. & Holroyd, K.A. A test of specific and non-specific effects in the biofeedback treatment of tension headache. *Journal of Consulting and Clinical Psychology*, 1980, *48*, 575-586.

Andreychuk, T. & Skriver, C. Hypnosis and biofeedback in the treatment of migraine headache. *International Journal of Clinical and Experimental Hypnosis*, 1975, *23*, 172-183.

Anthony, M. & Lance, J.W. The role of serotonin in migraine. In J. Pearce (Ed.), *Modern Topics in Psychiatry*, London: William Heinmann Medical Books Limited, 1975.

Appenzeller, O. Vasomotor function in migraine. *Headache* , 1969, *9*, 147-155.

Appenzeller, O., Davison, K. & Marshall, J. Reflex vasomotor abnormalities in the hands of migrainous subjects. *Journal of Neurological and Neurosurgical Psychiatry*, 1968, *26*, 447-450.

Appenzeller, O. Monaomines, headache and behavior. In O. Appenzeller (Ed.), *Pathogenesis and treatment of headache*. New York: Spectrum, 1976.

Ardlie, N.G., Glew, G. & Schwartz, C.J. Influence of catecholamines on nucleotide induced platelet aggregation. *Nature*, 1966, *212*, 415-417.

Bakal, D.A. & Kaganov, J.A. Muscle contraction and migraine headache: Psychophysiologic comparison. *Headache*, 1977, *17*, 208-215.

Bild, R. & Adams, H.E. Modification of migraine headaches by cephalic blood volume pulse and EMG biofeedback, *Journal of Consulting and Clinical Psychology*, 1980, *48*, 51-57.

Blanchard, E.B., Andrasik, F., Ahles, T.A., Teders, S.J. & O'Keefe, D.O. Migraine and tension headache: a meta-analytic review, *Behavior Therapy*, 1980, *11*,

613-631.

Blanchard, E.B. & Epstein, L.H. The clinical usefulness of biofeedback. In M. Hersen, R.M. Eisler and P.M. Miller (Eds.), *Progress in behavior modification*. New York: Academic Press, 1976.

Blanchard, E.B., Theobald, D.E., Williamson, D.A., Silver, B.V. & Brown, D.A. Temperature biofeedback in the treatment of migraine headaches. *Archives of General Psychiatry*, 1978, *35*, 581-588.

Budsynski, T.H. Biofeedback procedures in the clinic. In L. Birk (Ed.), *Behavioral Medicine*. New York: Grune & Stratton, 1973.

Budsynski, T.H., Stoyva, J.M., Adler, C.A. & Mullaney, D.J. EMG biofeedback and tension headache: A controlled outcome study. *Psychosomatic Medicine*, 1973, *35*, 484-496.

Cohen, M.J., Rickles, W.H. & McArthur, B.L. Evidence of physiological response stereotypy in migraine headache. *Psychosomatic Medicine*, 1953, *40*, 344-354.

Dalessio, D.J. (Ed.) *Wolff's headache and other head pain*. 3rd ed.; New York: Oxford University Press, 1972.

Dalessio, D.J. Migraine, platelets, and headache prophylaxis. *Journal of American Medical Association*, 1978, *239*, 52-53.

Deshmukh, S.V. & Meyer, J.S. Cyclic changes in platelet dynamics and the pathogenesis and prophylaxis of migraine. *Headache*, 1977, *17*, 101-108.

Downey, J.A. & Frewin, D.B. Vascular responses in the hands of patients suffering from migraine. *Journal of Neurological and Neurosurgical Psychiatry*, 1972, *35*, 258-263.

Elliot, K., Frewin, D.B. & Downey, J.A. Reflex vasomotor responses in the hands of patients suffering from migraine. *Headache*, 1973, *13*, 188-196.

Fahrion, S.L. Autogenic biofeedback treatment for migraine. In S. Karger (Ed.), *Research and clinical studies in headache*. North Caldwell, N.J. 1978.

Fair, P.L. Biofeedback strategies in psychotherapy. In J. Basmajian (Ed.), *Biofeedback Principles and Practice for Clinicians* . Baltimore: Waverly, 1979.

Fields, H.L. Secrets of the placebo. *Psychology Today*,

November, 1978.

Fanchamps, A. The role of humoral mediators in migraine headache. *Canadian Journal of Neurological Sciences*, 1974, *1*, 189-195.

Franks, J.D. *Persuasion and Healing*. Baltimore, John Hopkins Press, 1961

French, E.B., Lassers, B.W. & Desai, M.G. Reflex vasomotor responses in the hands of migrainous subjects. *Journal of Neurological and Neurosurgical Psychiatry*, 1967, *30*, 276-278.

Gellhorn, E. *Principles of autonomic-somatic integrations: Physiological basis and psychological and clinical implications*. Minneapolis: University of Minnesota Press, 1967.

Gellhorn, E. & Kiely, W.F. Mystical states of consciousness: Neurophysiological and clinical aspects. *The Journal of Nervous and Mental Disease*, 1972, *154*, 399-405.

Germana, J. Electromyography: Human and general. In R.F. Thompson and M.M. Patterson (Eds.), *Bioelectric recording techniques* (Part C). New York: Academic, 1974.

Green, E. & Green, A. General and specific applications of thermal biofeedback. In J. Basmajian (Ed.), *Biofeedback - Principles and practice for Clinicians*. Baltimore: Waverly, 1979.

Grings, W.W. & Dawson, M.E. *Emotions and Bodily Responses: A Psychophysiological Approach*. Academic Press, 1978.

Hackett, G. & Horan, J.J. Stress innoculation for pain: what's really going on? *Journal of Counseling Psychology*, 1980, *27*, 107-116.

Henryk-Gutt, R. & Rees, W.L. Psychological aspects of migraine. *Journal of Psychosomatic Research*, 1973, *17*, 141-153.

Hirsie, L.E. & Campbell, R.J. Psychiatric Dictionary (ed. 3). New York, Oxford University Press, 1960.

Hippocrates. *Hippocratic Writings*. Edited by G.E.R. Lloyd. Penguin Books, New York, 1978.

Hockaday, J.M., Macmillin, A.L. & Whitty, C.W.M. Vasomotor reflex response in idiopathic and hormone-dependent migraine. *Lancet*, 1967, *1*, 1023-1026.

Holroyd, K.A. Stress, coping, and the treatment of stress

related illness. In J.R. McNamara (Ed.), *Behavioral approaches in medicine: Application and analysis*. New York : Plenum Press, 1979.

Holroyd, K.A. & Andrasik, F. Coping and the self-control of chronic tension headache. *Journal of Consulting and Clinical Psychology*, 1978, *46*, 1036-1045.

Johnson, W.G. & Turin, A. Biofeedback treatment of migraine headache: A systematic case study. *Behavior Therapy*, 1975, *6*, 394-397.

Karlins, M. & Andrews, L.M. *Biofeedback: Turning on the power of the mind*. New York: Warner, 1972.

Kazdin, A.E. Non-specific treatment factors in psychotherapy outcome research. *Journal of Consulting and Clinical Psychology* , 1979, *47*, 846-851.

Kentsmith, D., Strider, F., Copenhaver, J. & Jacques, D. Effects of biofeedback upon suppression of migraine symptoms and plasma dopamine-B-Hydroxylase activity. *Headache*, 1976, *16*, 173-177.

Koenig, P. Lee's bilious pills: The placebo effect in patent medicine. *Psychology Today*, April, 1974.

Kudrow, L. Current aspects of migraine headache. *Psychosomatics*, 1978, *19*, 48-57.

Lacey, J.I., Bateman, D.E. & Van Lehn, R. Autonomic response specificity: An experimental study. *Psychosomatic Medicine*, 1953, *15*, 8-21.

Lacey, J.I. & Lacey, B.C. Verification and extension of the principle of autonomic response stereotypy. *American Journal of Psychology*, 1958, *71*, 50-73.

Lacey, J.I. & Lacey, B.C. The law of initial value in the longitudinal study of autonomic constitution: Reproducibility of autonomic responses and response patterns over a four year interval. *Annals of the New York Academy of Sciences*. 1962, *98*, 1257-1290.

Lake, A., Rainey, J., & Papsdorf, J.D. Biofeedback and rational-emotive therapy in the management of migraine headache. *Journal of Applied Behavior Analysis*, 1979, *122*, 127-140.

Lance, J.W. *The mechanism and management of headache* (2nd ed.). London: Butterworth, 1973.

Langley, L.L. *Physiology of man* (4th ed.). New York: Van Nostrand Reinhold, 1971.

Medina, J.L., Diamond, S., & Franklin, M.A. Biofeedback therapy for migraine. *Headache*, 1976, *16*, 115-118.

Melzak, R. *The Puzzle of Pain*. Penguin Books, Markham, Ontario. 1973.

Miller, N.E. Learning of visceral and glandular responses. *Science*, 1969, *163*, 434.

Mitch, P.S., McGrady, A. & Iannone, A. Autogenic feedback training in migraine: A treatment report. *Headache*, 1976, *15*, 267-270.

Morley, S. Migraine: A generalized vasomotor dysfunction? A critical review of the evidence. *Headache*, 1977, *17*, 71-74.

Mullinex, J.M., Norton, B.J., Hack, S. & Fishman, M.A. Skin temperature biofeedback and migraine. *Headache*, 1978, *17*, 242-244.

Peper, E. Frontiers of clinical biofeedback. In L. Birk (Ed.), *Biofeeback: Behavioral medicine*. New York: Grune & Stratton, 1973.

Pozniak-Patewicz, E. "Cephalgic" spasm of head and neck muscles. *Headache*, 1976, *16*, 261-266.

Price, K.P. Biofeedback and migraine. In R.J. Gatchel and K.P. Price (Eds.), *Clinical applications of biofeedback: Appraisal and status*. New York: Pergamon, 1979.

Price, K.P. & Tursky, B. Vascular reactivity of migraineurs and nonmigraineurs: A comparison of responses to self-control procedures. *Headache*, 1976, *16*, 210-217.

Roessler, R., Greenfield, N.S. & Alexander, A.A. Ego strength and response stereotypy. *Psychophysiology*, 1964, *1*, 142-150.

Sargent, J.D., Green, E.E. & Walters, E.D. The use of autogenic feedback training in a pilot study of migraine and tension headaches, *Headache*, 1972. *12*, 120-124.

Sargent, J.D., Green, E.E. & Walters, E.D. Preliminary report on the use of autogenic feedback training in the treatment of migraine and tension headaches. *Psychosomatic Medicine*, 1973, *35*, 129-135.

Sargent, J.D., Walters, E.D., & Green, E.E. Psychosomatic self-regulation of migraine headaches. *Seminars in Psychiatry*, 1973, *5*, 415-428.

Schultz, J.H. & Luthe, W. *Autogenic therapy*, vol. 1. New

York: Grune & Stratton, 1969.

Schwartz, G.E. & Weiss, S.M. Behavioral medicine revisited: An amended definition, *Journal of Behavioral Medicine*, 1978, *1*, 249-251.

Selby, G. & Lance, J.W. Observations on 500 cases of migraine and allied vascular headache. *Journal of Neurological and Neurosurgical Psychiatry*, 1960, *23*, 23-32.

Shapiro, A.K. Factors contributing to the placebo effect. Their implications for psychotherapy. *American Journal of Psychotherapy* , 1964, *73*, (Suppl).

Schultz, J.H. & Luthe, W. *Autogenic Therapy*, vol. 1. New York: Grune & Stratton, 1969.

Sicuteri, F. Headache as possible expression of deficiency of brain 5-hydroxytryptamine (central denervation supersensitivity). *Headache*, 1972, *12*, 69-72.

Silver, B.V., Blanchard, E.B., Williamson, D.A., Theobald, D.E. & Brown, D.A. Temperature biofeedback and relaxation training in the treatment of migraine headaches: One year follow-up. *Biofeedback and Self Regulation*, 1979, *4*, 359-366.

Sovak, M., Fronek, A., Helland, D.R. & Doyle, R. Effects of vasomotor changes in the upper extremities on the hemodynamics of the carotid arterial beds: A possible mechanism of biofeedback therapy of migraine (A preliminary report). In J.I. Martin (Ed.), *Proceedings of the San Diego Biomedical Symposium*, vol. 15. New York: Academic Press, 1976.

Sternback, R.A. The effects of instructional sets on autonomic responsivity. *Psychophysiology*, 1964, *1*, 67.

Stoyva, J. Why should muscular relaxation be clinically useful? Some data and 2 1/2 models. In J. Beatty and H. Legewie (Eds.), *Biofeedback and behavior*. New York: Plenum, 1977.

Stoyva, J. Guidelines in the training of general relaxation. In J. Basmajian (Ed.), *Biofeedback - Principles and practice for clinicians*. Baltimore: Waverly, 1979.

Stroebel, C.F. Practitioner's Manual to Accompany Quieting Response Training. B.M.A. Audio Cassette Programs, New York, 1978.

Stroebel, C.F. & Glueck, B.C. Biofeedback treatment in medicine and psychiatry: An ultimate placebo? *Seminars*

*in Psychiatry*, 1973, 5, 378-393.

Sullivan, E.A. *The future: Human ecology and education*. Homewood: ETC, 1975.

Surwit, R.S., Shapiro, D., & Feld, J.D. Digital temperature autoregulation and associated cardiovascular changes. *Psychophysiology*, 1976, *13*, 242-248.

Symon, L., Bull, J.W., duBoulay, E.P., Marshall, J. & Russell, R.W. Reactivity of cerebral vessels. In J.N. Cummings (Ed.), *Background to Migraine*, New York: Springer-Verlag, 1973.

Tunis, M.M. & Wolff, H.G. Studies on Headache. *Archives of Neurological Psychiatry*, 1953, *70*, 551-557.

Turin, A. & Johnson, W.G. Biofeedback therapy for migraine headaches. *Archives of General Psychiatry*, 1976, *33*, 517-519.

Waters, W.E. & O'Connor, P.J. Prevalence of migraine. *Journal of Neurological Neurosurgery in Psychiatry*, 1975, *38*, 613-616.

Weinstock, S.A. A tentative procedure for the control of pain: Migraine and tension headache. In D. Shapiro, T.X. Barber, L.V. DiCara, J. Kamiya, N.E. Miller & J. Stoyca (Eds.), *Biofeedback and self-control*, 1971, Chicago: Aldine, 1972.

Weiss, T. Weaning in biofeedback training. *American Journal of Psychiatry*, 1975, *132*, 1220.

Wickramasekera, I. Temperature feedback for the control of migraine. *Journal of Behavior Therapy and Experimental Psychiatry*, 1973, *4*, 343-345.

Wickramasekera, I. A conditioned response model of the placebo effect: Predictions from the model, *Biofeedback and Self-Regulation*, 1980, *5*, 1-10.

Winer, B.J. *Statistical Principles in Experimental Designs*. McGraw-Hill, 1962.

Yates, A. *Biofeedback and the Modification of Behavior*. New York: Plenum Press, 1980.

# APPENDIX A

## *TREATMENT CONTRACT*

*Participant*

(A) I understand that my participation in the migraine treatment program will require my full cooperation and consent in each of the following components of the study:

1) Punctual attendance at all 8 treatment sessions scheduled twice per week over 4 weeks;

2) Attendance at one pretraining physiological monitoring session held several days before treatment, and one posttreatment follow-up session held two months after the treatment period;

3) Twice daily 15 minute home practice and monitoring of specific relaxation skills learned in treatment to continue throughout the 4 week training period and two month follow-up;

4) Hourly monitoring of headache activity and medication consumption during the weeks before treatment, the weeks of treatment, and one month after treatment;

5) Keeping a headache diary for one year following the treatment period;

6) Obtaining proof of recent medical examination by a physician;

7) Payment of $40 fee to cover the cost of equipment maintenance and the materials used in this research;

8) Notifying Patrick Carney, C/O the Department of Educational Psychology, 6th floor - Education North, University of Alberta should my address change.

(B) I acknowledge and agree that neither the University or the treatment staff shall be responsible for loss of or damage to any personal property incurred in the course of this treatment project.

Date: Signature:

Signature of Witness:

*Therapist*

(C) I promise that all records of Participants' names, addresses, and personal information will be kept confidential. At the completion of this study a summary of the results obtained shall be made available on request to all those who fully participate.

Date: Signature:

# APPENDIX B

## *MEDICAL FORM*

Name of Physician:

Name of Patient:

Address:

Date of Birth:
Address:

Phone:

The above named patient has been selected to participate in a treatment program for headache patients being conducted at the University of Alberta, Department of Educational Psychology. This research is being supervised by Dr. George Fitzsimmons. The treatment being used will involve relaxation training and psychophysiological monitoring including electromyography, galvanic skin response, and surface skin temperature.

We are requesting each patient to obtain the signature of a physician to varify that they have received a recent medical examination and to ensure that there is no medical reason why they should not participate in the research project.

*For Physician*

(A) This is to certify that has been medically examined and I do not advise against his/her participation in the program described.

(B) I (do, do not) agree that the headache pain which this person reports is of the migraine form.

Date: Physician's Signature:

# APPENDIX C

## *PROCEDURE FOR CONDUCTING STRESS PROFILE*

Record ambient temperature

Seat client in recliner chair and tilt chair to the first reclined position. Inquire whether headache is now present and if so reschedule the session.

Attach biofeedback instrumentation.

Read the following instructions to the subject:

> "Today's session will last approximately 30 minutes. What I am goin to do is attach you to three biofeedback instruments in order to see what levels of activity you produce in three different physiological systems: (a) skin temperature, (b) muscle tension, and (c) skin perspiration. These instruments will not shock you or harm you in any way, they merely attach on to the surface of your skin with these wires. We are hooking you up today in order to find out how your body activity corresponds to your headache pattern, and how the relaxation treatment program changes both your body activity and your headache pattern. Do you have any questions?...
>
> "For the next 15 minutes I would like you to relax comfortably with your eays open and just listen to the music being played in the background. Try to avoid unpleasant thoughts and just enjoy this 15 minutes of rest. After 15 minutes have elapsed I will ask you to sit for about 10 minutes with your eyes closed. Please try to sit quietly without looking around or talking, and try to keep your hands still on the arm rest with your palms facing upward. Do you have any questions?... Okay then, starting with your eyes open, just relax and I will tell you when 15 minutes are up."

Begin monitoring. Continue for 15 minutes.

After 15 minutes say:

> "And now I would like you to sit for about 10 minutes with your eyes closed. Try not to fall asleep."

After three minutes say:

"OKay, while keeping your eyes closed now I want you to perform a mental task for me. I want you to subtract seven from 1000 and then to continue subtracting seven from your answers as fast as possible until I tell you to stop. Do this in your head not out loud. OKay so 1000 minus seven is ... (pause), now keep going to yourself."

After three minutes say "stop." Ask, "what number did you get to?" Say to subject: "Now I just want you to relax with your eyes closed and listen to the music without interuption for five minutes and then we are finished.

After five minutes end the session and disconnect the instrumentation from the client.

# APPENDIX D

## *EMG TRAINING PROCEDURES AND INSTRUCTIONS - SESSIONS 1 TO 8*

1. Attach the biofeedback instrumentation

2. Read the following rationale to the subject:

> "The eight treatment session you are receiving are designed to teach you how to produce more effective physiological relaxation at will. Your final goal in treatment is to become able to discriminate excessive stress in your body and be able to remove such stress in order to prevent migraine headaches. Regular and consistent practice at removing excessive stress will eventually develop into a life-style habit. When this occurs your body will maintain a more relaxed level of arousal without conscious effort. It may take somewhere between a couple of weeks to several months to develop this automatic habit, depending upon the amount of relaxation practice you do and the strength of the stress habit you now have.
>
> In biofeedback training you will learn to relax efficiently, guided by the feedback signal. The idea is to slow down the clicking noise which indicates the level of muscle tension in your head region. Slower clicking means less tension. Over time you will learn to produce lower levels of tension in less time and to maintain these low levels for longer periods. Even though the biofeedback is only attached to the head region it is to your advantage to learn to relax throughout your entire body.
>
> Biofeedback guided relaxation takes place in 3 stages. The first stage is called the "awareness" stage where your brain is merely made aware of how much clicking feedback corresponds to how much muscle tension. Gradually the second stage emerges where in addition to becoming aware of tension levels you become able to control the tension and further reduce it. This second stage is known as the "control" stage.
>
> Please note that the control stage takes time to emerge because you must learn the skill involved. Also note that contrary to most other intentional learning you do, learning to relax does not involve active striving. The more you strive the more tense you will become. Instead of actively striving to

reduce muscle tension you must passively concentrate on the feedback signal and "allow" the clicking to reduce. In other words, "let it happen."

The final stage of biofeedback guided relaxation, following awareness and control is the "weaning" stage. Weaning involves practice at producing the relaxation response in the absense of the biofeedback signal (clicks). Such practice will be provided in sessions 5, 6, 7, and 8. In this way you can learn an effective relaxation skill which is not dependent upon biofeedback.

Many persons have asked what thinking strategies they should be using to slow the clicking as they passively concentrate. Other than advising such persons to avoid unpleasant thoughts or stress-related ruminations, there is no particular strategy that everyone will find effective. Some people use mental images of relaxing settings such as laying on a warm beach, skiing down a mountain in slow motion, or watching a beautiful sunset. Others think suggestive phrases to themselves such as "I am becoming more relaxed, more calm and more quiet, I am becoming warm and relaxed." Others do not think about anything, they let their minds go blank. Most people find some particular strategy useful at first but as they learn to relax efficiently, letting go of tension becomes a skill they can utilize without any conscious strategy. Over the course of the 8 training sessions, I would like you to use whatever strategies you feel comfortable with to relax. But remember, the important thing is not to force any approach or to try too hard, because effort is the opposite of relaxation. Just let the approach you choose flow, just imagine it is already happening."

3. In Session 1 give the following instructions:

"The relaxation session will last 20 minutes. During biofeedback please practice your relaxation with your eyes closed as much as possible. I will go over the tension level results obtained with you today at the end of the session. Do you have any questions?... Now you may begin."

In Sessions 2 to 4 give the following instructions.

"The relaxation session will last 20 minutes. During biofeedback please practice your relaxation with your eyes closed for five minutes and then with your eyes open for 15 minutes. When I tell you that five minutes are up please open your eyes slowly and try to maintain low tension as you watch the E.M.G.

feedback gauge."

In Sessions 5 to 8 give the following instructions:

"The relaxation session will last 20 minutes. During biofeedback please practice your relaxation with your eyes closed for five minutes and then with your eyes open for 15 minutes. During the final five minutes I will turn off the visual and sound feedback so that you can practice your skills without the biofeedback.

4. Conduct biofeedback training.

5. At the end of each session give the following instructions:

"Please spend 5 minutes writing down a description of the strategies which you employed to relax and also identify any feelings or sensations which appeared to be associated with slower clicking. A new summary will be written each session and taken home with you until the next session at which time we would like you to hand it in for our records. Go ahead now, I will tell you when 5 minutes have elapsed."

After the 5 minute period give the following instructions:

"For training to be effective in suppressing migraines you must practice relaxation twice daily for 15 minutes utilizing the strategies you have written down. In this manner you will be attempting to duplicate outside the lab. the same feeling state associated with slow clicking biofeedback. Please remember to monitor the total minutes of daily relaxation you practice on your headache monitoring forms. Ideally you should schedule your relaxation practice periods in a manner which will break up your daily stress cycle"

6. Discuss the subject's progress during the session with him. Show him the minute-to-minute E.M.G. levels that he achieved and point out how ideally he will be learning how to become more relaxed faster, and be able to maintain such relaxed levels longer.

7. After each treatment sessson discuss medication consumption with the subject. Subjects should be advised and repeatedly reminded to monitor their medication intake and to consult with their physicians about any changes required in their prescriptions. Inform subjects that increased relaxation may alter the effects of their

medication, migraine or otherwise. This is especially true for subjects taking medication for hypertension, or diabetes.

# APPENDIX E

## *TEMPERATURE TRAINING PROCEDURES AND INSTRUCTIONS - SESSIONS 1 to 8*

1. Attach biofeedback instrumentation.

2. Read the following rational to the subject:

> "The eight treatment sessions you are receiving are designed to teach you how to produce more effective physiological relaxation at will. Your final goal in treatment is to become able to discriminate excessive stress in your body and be able to remove such stress in order to prevent migraine headaches. Regular and consistent practice at removing excessive stress will eventually develop into a life-style habit. When this occurs your body will maintain a more relaxed level of arousal without conscious effort. It may take somewhere between a couple of weeks to several months to develop this automatic habit, depending upon the amount of relaxation practice you do and the strength of the stress habit you now have.
>
> In biofeedback training you will learn to relax efficiently, guided by the feedback signal. The idea is to warm your hands voluntarily as you relax and learn how to use hand warming as an index of your relaxation level. Over time you will learn how to produce greater levels of relaxation in less time and to maintain these levels for longer periods. Even though the biofeedback is only attached to one of your hands it is to your advantage to learn how to hand warm as part of a total body relaxation response.
>
> Biofeedback guided relaxation takes place in 3 stages. The first stage is called the "awareness: stage where your brain is merely made aware of how temperature changes correspond to vascular changes brought about by stress and relaxation. Gradually the second stage emerges where in addition to becoming aware of stress levels you become able to control the stress and further reduce it. This second stage is known as the "control" stage.
>
> Please note that the control stage takes time to emerge because you must learn the skill involved. Also note that contrary to most other intentional learning you do, learning to relax does not involve

active striving. The more you strive the more stressed you will become. Instead of actively striving to warm your hands and relax you must passively concentrate on the feedback signal and "allow" the warming to occur. In other words, "let it happen."

The final stage of biofeedback guided relaxation awareness and control is the "weaning" stage. Weaning involves practice at producing the relaxation response in the absence of the biofeedback signal (temperature gauge). Such practice will be provided in sessions 5, 6, 7 and 8. In this way you can learn an effective relaxation skill which is not dependent upon biofeedback.

Many persons have asked what thinking strategies they should be using to induce hand warming as they passively concentrate. Other than advising such persons to avoid unpleasant thoughts or stress-related ruminations there is no particular strategy that everyone will find effective. Some people use mental images of relaxing, settings such as laying on a warm beach, skiing down a mountain in slow motion or watching a beautiful sunset; other think suggestive phrases to themselves such as "I am becoming more relaxed, more calm and more quiet, I am becoming warm and relaxed"; others do not think about anything, they let their minds go blank. Most people find some particular strategy useful at first but as they learn to relax efficiently, letting go of stress becomes a skill they can utilize without any conscious strategy.

Over the course of the 8 training sessions I would like you to use whatever strategies you feel comfortable with to relax. But remember the important thing is not to force any approach or to try too hard, because effort is the opposite of relaxation. Just let the approach you choose flow, just imagine it is already happening."

3. In Sessions 1 to 4 give the following instructions:

"The relaxation session will last 20 minutes. The more you relax the more your hand temperature will increase up to a maximum of 90-96 degrees. I will go over the temperature relaxation results obtained with you today at the end of the session. Do you have any questions?... Now you may begin. Please practice with your eyes open."

In Sessions 5 to 8 also say:

"During the final five minutes I will discontinue feedback so that you can practice relaxation in its absence.

4. Conduct biofeedback training.

5. At the end of each session, give the following instructions:

"Please spend five minutes writing down a description of the *strategies* which you employed to relax and also identify any *feelings* or *sensations* which appeared to be associated with hand warmings. A new summary will be written each session and taken home with you until the next session at which time we would like you to hand it in for our records. Go ahead now, I will tell you when 5 minutes have elapsed."

After the 5 minute period give the following instructions: "For training to be effective in suppressing migraines you must practise relaxation twice daily for 15 minutes utilizing the strategies you have written down. In this manner you will be attempting to duplicate outside the lab. the same feeling state associated with hand warming biofeedback in the lab. Please remember to monitor the total minutes of daily relaxation you practice on your headache monitoring forms. Ideally, you should schedule your relaxation practice periods in a manner which will break up your daily stress cycle.

6. Discuss the subject's progress during the session with him. Show him the minute-to-minute temperature levels that he achieved and point out how ideally, he will be learning how to become more relaxed, faster, and be able to maintain such relaxed levels longer.

7. After each treatment session discuss medication consumption with the subject. Subjects should be advised and repeatedly reminded to monitor their medication intake and to consult with their physicians about any changes required in their prescriptions. Inform subjects that increased relaxation may alter the effects of their medication, migraine or otherwise. This is especially true for subjects taking medication for hypertension, or diabetes.

8. In Session 2 (Session 3 for Combined treatment) provide subjects with Biotic Bands and instructions (see Appendix F). Ask subjects to practice once a day for 15 minutes and to monitor temperatures as instructed.

# APPENDIX F

## *BIOTIC BAND MONITORING AND RECORDING*

Please use your Biotic Band device to monitor finger temperature for one 15-minute relaxation session per day. Attach the band to the middle finger of your non-dominant hand. Place the band with the temperature scale on the palmar surface of your finger, and centre it mid-way along the length of your finger. The band should be snug but not tight. While relaxing try to sit in a comfortable chair with arm rests so that your hand temperature will not be effected by warmth from your lap.

As you practice relaxation note how your finger temperature increases. On your headache monitoring form, write down your finger temperature (a) after the band has been on your finger for 1/2 minute and (b) after exactly 15 minutes of relaxation (e.g. (a) 84, (b) 91).

Please avoid crusing or crumpling the band as they may become inaccurate with abuse. If you think that your band has broken bring it in to your next training session. We would appreciate having the bands returned at the end of treatment.

BIOTIC-BAND II has a range of 20.0°F divided into two degree intervals which are indicated on the band by the printed numbers. The liquid crystal squares beside the numbers light up when the temperature of the finger being monitored comes within that two degree range. Within each range of two degrees, color changes indicate smaller changes in the temperature. Each color change equals a change of 0.5°F as shown in the table below.

| Lighted Degree | Red-Tan | Orange | Yellow-Green | Blue-Green | Blue |
|---|---|---|---|---|---|
| 78° | 78° | 78.5° | 79° | 79.5° | 80° |
| 80° | 80° | 80.5° | 81° | 81.5° | 82° |
| 82° | 82° | 82.5° | 83° | 83.5° | 84° |

| | | | | | |
|---|---|---|---|---|---|
| 84° | 84° | 84.5° | 85° | 85.5° | 86° |
| 86° | 86° | 86.5° | 87° | 87.5° | 88° |
| 88° | 88° | 88.5° | 89° | 89.5° | 90° |
| 90° | 90° | 90.5° | 91° | 91.5° | 92° |
| 92° | 92° | 92.5° | 93° | 93.5° | 94° |
| 94° | 94° | 94.5° | 95° | 95.5° | 96° |
| 96° | 96° | 96.5° | 97° | 97.5° | 98° |

In taking a reading *always read the highest temperature showing*. The *purple color* which may sometimes be visible on some squares should always be *ignored*.

# APPENDIX G

## *SUMMARIES OF ANALYSES OF VARIANCE*

Summary of Analysis of Variance for Headache Intensity Variable, Reactivity Grouping, Three-Factor Repeated Measures, (N=48).

| Source | Sums of Squares | Degrees of Freedom | Mean Squares | F | P |
|---|---|---|---|---|---|
| Between Subjects | 5604.64 | 47 | | | |
| A | 88.53 | 1 | 88.53 | 0.73 | 0.40 |
| B | 279.41 | 2 | 148.70 | 1.22 | 0.30 |
| AB | 113.36 | 2 | 56.68 | 0.47 | 0.63 |
| Subjects within Groups | 5105.34 | 42 | 121.56 | | |
| Within Subjects | 1183.90 | 96 | | | |
| C | 265.52 | 2 | 132.76 | 13.29 | 0.000* |
| AC | 1.71 | 2 | 0.86 | 0.09 | 0.92 |
| BC | 21.23 | 4 | 5.31 | 0.53 | 0.71 |
| ABC | 56.62 | 4 | 14.15 | 1.42 | 0.24 |
| C x Subjects within Groups | 838.82 | 84 | 9.99 | | |

* Conservative F (1,42) = 0.001
(see Winer P. 523)

Summary of Analysis of Variance for Headache Intensity Variable, Recovery Grouping, Two-Factor ANOVA, (N=55).

| Source | Sums of Squares | Degrees of Freedom | Mean Squares | F | P |
|---|---|---|---|---|---|
| A | 86.11 | 1 | 86.11 | 2.10 | 0.15 |
| B | 82.93 | 2 | 41.46 | 1.01 | 0.37 |
| AB | 320.82 | 2 | 160.41 | 3.92 | 0.03 |
| ERRORS | 2005.32 | 49 | 40.92 | | |

Summary of Analysis of Variance for EMG Training Data, Reactivity Grouping, Three-Factor Repeated Measures (N=48).

| Source | Sums of Squares | Degrees of Freedom | Mean Squares | F | P |
|---|---|---|---|---|---|
| Between Subjects | 303.11 | 47 | | | |
| A | 1.06 | 1 | 1.06 | 0.15 | 0.70 |
| B | 3.30 | 2 | 1.65 | 0.24 | 0.79 |
| AB | 10.29 | 2 | 5.14 | 0.75 | 0.48 |
| Subjects within Groups | 288.47 | 42 | 6.87 | | |
| Within Subjects | 566.10 | 480 | | | |
| C | 147.43 | 10 | 14.74 | 16.83 | 0.00* |
| AC | 6.51 | 10 | 0.65 | 0.74 | 0.68 |
| BC | 29.02 | 20 | 1.45 | 1.66 | 0.04** |
| ABC | 15.14 | 20 | 0.76 | 0.86 | 0.63 |
| C x Subjects within Groups | 368.00 | 420 | 0.88 | | |

* Conservative F (1,42) = 0.001
** Conservative F (1,42) = 0.250
(see Winer P. 523)

Summary of Analysis of Variance for EMG Training Data.

Recovery Grouping, Two-Factor ANOVA, (N=55).

| Source | Sums of Squares | Degrees of Freedom | Mean Squares | F | P |
|---|---|---|---|---|---|
| A | 8.66 | 1 | 8.66 | 3.24 | 0.08 |
| B | 4.54 | 2 | 2.27 | .85 | 0.43 |
| AB | 3.39 | 2 | 1.70 | .64 | 0.64 |
| ERRORS | 130.88 | 49 | 2.67 | | |